

Differential Diagnosis of **CHEST DISEASES**

by

JACOB JESSE SINGER, M.D. F.A.C.P. F.C.C.P.

*Medical Director of the Rose Landy and Craft Foundation, Beverly Hills, California; in
Chest Diseases, Clinics of Lebanon Hospital, Los Angeles, California; formerly
Associate Professor of Clinical Medicine, Washington University
(St. Louis, Missouri) and University of Southern
California (Los Angeles, California)*

WITH 111 ILLUSTRATIONS

LONDON
HENRY KIMPTON
25, BLOOMSBURY WAY, W.C.1

ALL RIGHTS RESERVED, F

UNIVERSITY OF AMERICA

DEDICATED
to the Memory of
ROSL LAMPERT GRAFF

PREFACE

NEARLY every literary effort is undertaken in the conviction that a real need exists for the work the author conceives. In spite of the plethora of

and coherently concluding with a section suggesting a basis of differentiation between the abnormality discussed and other abnormalities with which it might be confused.

Röntgenology is a useful auxiliary instrument the diagnostician of today has at his disposal but it is not by itself a basis for final diagnosis. The determination of any specific abnormality is still dependent upon the analysis and correlation of data from many sources. This has been often said. I

story.

The diagnostic procedure is much like that of the detective story. The various findings are clues thrown off from a basic and unified process. These clues are correlated and made relevant by an understanding of the likely or possible processes. Hence the emphasis on etiology in pathology.

In style the aim has been for conciseness. Too often a reader of medical literature is lost in a demonstration of academic proficiency—the usually entirely irrelevant and perfunctory outline of medical history—and a maze of conflicting quoted opinions. Compilations of other investigations and case reports are most useful and are entirely legitimate but they comprise a separate field of medical writing to be distinguished from a book of this type.

I have indicated my opinion at all times. I have discussed the various pathologic processes in terms of my conclusions. I accept the almost in-

a certainty of majority. I have tried to indicate the opposing points of view. It is unfortunately not possible to develop each subject thoroughly in a single volume.

In rhetoric I have sought simplicity. The need for an extensive technical vocabulary is of course undisputed but the attempt to coin new words should be restrained. It is a regrettable but undeniable habit of the human mind including the medical mind to create new terms. Each specialty has its own terms which should be known. Superfluous terminology is frequently a disadvantage.

PREFACE

These then have been my objectives—correct diagnosis regarded as the most important attribute on disease consciousness and simplicity. My primary audience is the general practitioner that vital and neglected figure in medicine upon whom in the last analysis progress in the conquest of disease largely depends. It is to be hoped of course that the book will not be uninteresting to the specialist in chest diseases and to roentgenologists.

Many thanks are due to Dr. Morris Horwitz radiologist for his help in obtaining roentgen ray films from the Los Angeles General Hospital and his private practice and also for his many invaluable suggestions. Dr. Leonard Asher Dr. Alfred Goldman Dr. Jacob Segal Dr. Seymour Furber and Louis Spencer Levy all of California have also been extremely helpful. I want to acknowledge the editorial assistance of William Brindt a grammarian whose aid in rearranging my materials contributed to the correctness that I hope this book incorporates. I also wish to extend my personal thanks to my secretary Helen Shinn who has been devoted to her work at all times. To the Rose Lampert Gruff Foundation my sincerest thanks for contributing both financial and other help to aid in presenting this book to the profession. Lastly I wish to acknowledge the understating assistance extended by the publishers Lecky and Lebigue.

Beverly Hills California

JACOB JESSE SINGER M.D.

CONTENTS

CHAPTER	DIAGNOSTIC METHODS	PAGE
1	HISTORY AND PHYSICAL EXAMINATION	9
2	LABORATORY METHODS	17
3	MECHANICAL AIDS	23
4	RADIOLOGY	30

THORACIC CAGE

5	DEFORMITIES, FRACTURES AND INFLAMMATIONS OF THE THORACIC CAGE	39
6	CERVICAL RIB	45
7	TUMORS OF THE CHEST WALL	48

DISEASES OF THE PLEURA

8	ANATOMY AND MISCELLANEOUS DISEASES	55
9	ACUTE FIBRINOUS PLEURISY	61
10	SEROFIBRINOUS PLEURISY	64
11	HYDROTHORAX	74
12	EMPYEMA (SUPPURATIVE PLEURISY)	76
13	PUTRID EMPYEMA	83
14	TUBERCULOUS EMPYEMA	85
15	SPONTANEOUS PNEUMOTHORAX	88

MEDIASTINUM

16	ANATOMY	97
17	MEDIASTINITIS	100
18	TUMORS OF THE MEDIASTINUM	106

DIAPHRAGM

19	ANATOMY AND EVENTRATION OF THE DIAPHRAGM	125
20	DIAPHRAGMATIC HERNIA	131
21	SUBPHRENIC ABSCESS	136

TRACHEO-BRONCHIAL TREE AND LUNGS

PAGE

22	ANATOMY AND MISCELLANEOUS DISEASES	141
23	BRONCHIECTASIS	152
24	BRONCHOPNEUMONIA	169
25	LOBAR PNEUMONIA	175
26	LIPOID PNEUMONIA	184
27	VIRUS (ATYPICAL) PNEUMONIA	192
28	EOSINOPHILIC PNEUMONIA	199
29	PULMONARY ABSCESS	202
30	PNEUMOCONIOSIS	215
31	PULMONARY MICOSIS	226
32	HODGKIN'S DISEASE	239
33	PULMONARY EMPHYSEMA	243
34	NON-PARASITIC CYSTS	251
35	HYDATID CYSTS	258
36	TUBERCULOSIS—MEDICAL ASPECTS	266
37	TUBERCULOSIS—SURGICAL ASPECTS	285
38	BENIGN TUMORS OF THE LUNG	310
39	CANCER OF THE LUNG (CARCINOMA)	316
40	SARCOIDOSIS	330
	INDEX	335

DIAGNOSTIC METHODS

I. HISTORY AND PHYSICAL EXAMINATION

THE presence of a pathologic condition in the chest is fairly easy to discover the diagnosis of the specific disease is quite another matter. In spite of the improvement in the roentgen ray procedures the mechanical aids and the new laboratory techniques such diagnosis of chest diseases is largely a matter of judgment. There are few symptoms that indicate a

pose of this book is to present to the physician as specifically as possible the important diseases of the chest and to follow with case histories of some of them that indicate the method of diagnosis. The method that must be used in the chest is comparative—the relation of tuberculosis to carcinoma their similarities and differences.

The author thinks it of benefit to preface this chief section of the book

author's experience indicates are important and yet sometimes overlooked. It is designed for the general practitioner and is intended to indicate with out elaboration the wide variety of sources of information that should be considered in a chest diagnosis.

HISTORY

A history as complete as possible is of course important to any diagnosis and it can be particularly helpful in the diagnosis of suspected chest abnormality. However one must remember that it is the least objective evidence of the examination. Individuals occasionally unintentionally suppress important information as to symptoms to prevent discovery of a condition they suspect to be serious. Malingerers give purely fictional histories. Even without these hazards the history is distorted by the fact that it depends on human memory and its reliability is further decreased because it is interpreted to a degree by even the most honest patient. A history can be useful in determining the duration of a disease and to some extent the nature of the disease.

HISTORY AND PHYSICAL EXAMINATION

pulmonary diseases are unfortunately similar coughing with or without expectoration hemoptysis persistent loss of weight and strength elevation of temperature pain in the chest and night sweats A family history of tuberculosis may or may not be correct As in case histories a family history is most useful when its indications are confirmed by physical studies

INSPECTION

Patients should be examined in a good light with the chest completely uncovered. Light can be too bright as well as too dim small masses under the skin that are readily observable in the right light can be obscured by too much light Metastases may appear in any region of the skin due to primary malignancy elsewhere on examination should be complete The examiner should note the facial expression the color of the skin the state of nutrition and the elasticity or flabbiness of the muscles Sometimes even the positions the patient assumes in the course of the examination are significant to the diagnosis

Inspection of the Eyes — Pupils should be examined for irregularities in size and shape. Unequal pupils are not unusual in pathologic states of the lung with contraction ordinarily noted on the side of the affected lung. This condition may be due to pressure on the sympathetic nerves by mediastinal tumors or by the road masses. Irregular and fixed pupils may indicate syphilis. Proptthalmus and ptosis of the upper lid slight elevation of the lower lid and constriction of the pupils (Horner's syndrome) are caused by paralysis of the cervical sympathetic nerve. Exophthalmus is frequently found in hyperthyroid diseases.

Inspection of the Nose — Inspection of the nasal passage frequently affords important clues to the diagnosis of chest conditions. One should look for polyps either tumorous or due to hypertrophy of the membrane deflected septum and evidence of acute or chronic sinus infection. Syphilitic ulcerations and perforations of the nasal septum may easily be seen. Frosions of the mucous membrane lining may show evidences of bleeding which had previously been thought to come from the lungs. The nasal cavity is oftentimes a source of infection in lung diseases. Furtulent discharge from purulent sinus infections may be aspirated into the lungs and produce abscesses or may light up low grade infections already present. A relationship between nasal infection and pulmonary suppurations in the same individual seems to be almost invariable and the treatment of the former is almost always of considerable benefit to the pulmonary condition.

Any infection in the nasal passages should be further investigated with roentgen ray studies of the sinuses.

Inspection of the Mouth and Pharynx — There is a close relation between pathologic conditions in the mouth and pharynx and abnormal conditions in the lungs in many instances the oral cavity is the source of lung infection. Enlarged tonsil and lymphoid masses in the posterior pharynx are particularly associated with lung disease. The tonsillar pillars should always be retracted for a direct view of the tonsils since an almost symptomless disease process in the crypts is frequently the source of lung

infection. Hutchinson's teeth is evidence of syphilis, dental caries and pyorrhea have also been indicated as possible sources of pulmonary infection. Pyorrhea is frequently found in association with lung abscess. In all cases of gingivitis the secretion should be studied for the presence of *Spirochetes*.

The examiner should also be alert for discoloration and lesions that

tions. On the other hand the examiner must remember that toxemia resulting from infection in the tonsils and around the mouth frequently produces symptoms not unlike those of chronic pulmonary suppurative infections.

It is advisable that all physicians interested in pulmonary diseases be thoroughly familiar with laryngoscopic appearances. Both direct inspection with the laryngeal mirror and by means of the laryngoscope are available to the examiner. It is possible to see small tumors on the vocal chords or deformities of the chords. The paralysis of one chord is very noticeable and particularly significant to diagnosis since such paralysis is frequently caused by tumors within the lung or mediastinum. Tuberculosis malignancy and syphilitic ulcers can be seen with ease but the true character of such ulceration must be determined by biopsy and microscopic studies of the secretions and tissue.

The examination of a large part of the tracheobronchial tree must be made bronchoscopically.

Inspection of the Neck. This area offers several readily observable symptoms of pathologic chest conditions. Enlargement of the thyroid can be easily noted. Although spasticities and deformities of the neck when caused by torticollis are usually due to spasm or injury to the sternocleidomastoid muscle they can also be caused by enlarged lymph nodes or by tumors of the tonsils, neck or pharynx. Similar symptoms have also been found to be caused by the presence of a cervical rib.

Cervical nodes are frequently seen as well as palpated and are a clue to possible tuberculosis.

Usually not

there is any secretion. If there is it should be checked for bacterial and fungus content.

Cyanosis and edema of the face and neck are occasionally found. They may be produced by any obstruction to the superior vena cava but are usually due to mediastinal masses of Hodgkin's disease or neoplasm.

Inspection of the Chest.—The chest should be closely examined from all views— anterior, posterior and lateral. The shape of the chest as a whole should be observed. Deformities of the thoracic cage are easily seen and may be caused by deformity of the spine, by post-operative changes due to thoracoplasty operations and occasionally by traumatic damage to the rib cage. Severe cases of pigeon breast (Pectus Carinatum) or funnel

chest (Pectus Excavatum Trichterbust) may require appropriate treatment

Unequal movement between the two sides of the chest may suggest tuberculosis of the lungs or pleura but a wide variety of abnormalities may cause this symptom Delayed expansion on inspiration suggests the possibilities of adhesions or fluid within the chest sometimes it is caused by a tumor or pneumonic consolidation

A narrow
culosis of
intercosta
fluid in th

indicated by masses between the ribs sometimes the ribs are distorted by them Abnormal depressions above and below the clavicle are important Notching of the ribs is frequently seen in instances of coarctation of the aorta

A hernia of the lung particularly at the apex can be noted in a visual

piration upon the lower side of the thorax when the patient is lying down

The axillary areas should be closely examined with the patient's arms above his head for nodes or evidences of irritation or infection

Other the
chest are rev
pilo ste
omata chondromata—and the malignant tumors primary sarcoma and
carcinoma

cervical and thoracic but the examiner can sometimes find important symptoms in other regions Distention or contraction of the abdomen is sometimes present large and tortuous veins or irregular masses extending upward through the abdomen are not uncommon Edema of the skin and anasarca can also be noted

The inguinal region should be checked for inguinal hernia sometimes

visible when the patient coughs. Enlarge lymph nodes and sinuses should also be watched for

The extremities should also be observed for metastatic tumor masses and cutaneous fungous lesions. Edema may be noted in pulmonary conditions that lead to cardiac dysfunction.

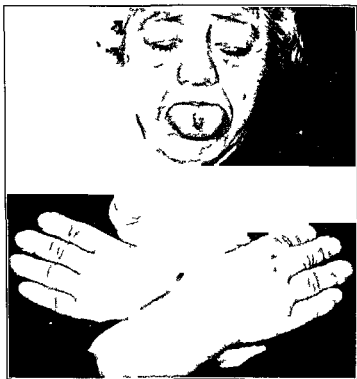


Fig. 1. Photograph of patient with redundant eyelids, macroglossia, clubbed fingers and dermatism. (B. M. Fred. Courtesy of Arch. Int. Med.)
Diagnosis: Osteoarthritis

PALPATION

Palpation is useful as an auxiliary to inspection and other techniques of diagnosis; it also reveals certain definite information not obtainable by any other method.

It is the only effective means of examining the deep lymphatic nodes of the neck, the supraclavicular nodes, the occipital and cubital nodes, and the cervical nodes. All these areas should be carefully examined; even a slight swelling may be important to diagnosis.

By palpation the examiner can determine tumor masses as solid or fluctuant. A mass of nodes can be distinguished as discrete or conglomerate. Abnormal tension in the muscles overlying the lungs should be especially noted. This latter symptom indicates only inflammation and diseased lung tissue and is not a specific symptom of tuberculosis as is sometime thought.

The presence of masses in the abdomen can be detected, and careful palpation can reveal irregularities in the position, size and shape of the liver, spleen and kidneys. The cardiac pulsations, the location of the apex beat, thrills of aneurisms and heavy pulsations of the carotids are all important conditions that may be determined by palpation. The intercostal spaces should be examined for pathologic conditions such as neurofibroma of the intercostal nerves.

This is also the chief method for locating points of spasticity and tenderness. It is noteworthy in this connection to recall however how frequently the patient will give evidence of tenderness over the appendix region when *there is inflammation of the pleura or pneumonia in the right chest.* It is therefore necessary that caution be exercised in the interpretation of the location of the diseased area. Tenderness in the intercostal area is perhaps more reliable as an indication of the location of suppurative conditions than tenderness elsewhere but even in that location it is still possible that the pain is reflex.

The interpretation of tactile fremitus requires some experience but this palpation of voice and breath sounds can be very helpful to visualize the state of the viscera. By this method the physician can determine with some accuracy the presence or absence of fluid, solid tissue, cavitation or emphysema.

THE APPRECIATION OF SOUND

The lungs are beyond our direct vision and palpation, the examiner's problem is to visualize them through indirect methods. The foregoing

diagnosis possible

Percussion is the technique of the production of sounds by the impact

examiner in using percussion on the lungs measures the difference aurally between the characteristic resonant tone of the lung itself (as an air filled body) and the tone of areas where abnormal conditions mute or modify it. A skilled practitioner can not only detect lesions of the lung by this method

but can determine in many instances their shape and extent. Even rather small cavities will be suspected by the increased resonance of the chest wall over them. Percussion cannot, however, be used to detect deep lesions which do not appreciably modify chest wall resonance.

Auscultation is the detection by ear of sound produced within the body. Chest auscultation is directed to sounds produced by the interior movement of air, breath, voice, etc., which are audible on the chest wall alone. These sounds are weak and diffused and can only be detected when the ear is immediately against the chest or with the aid of the stethoscope. The variation in these sounds is helpful in determining general conditions, but auscultation must be used in conjunction with percussion for exact localization. The examiner must be careful to eliminate extraneous sound as much as possible. All the chest auscultation sounds should be considered in a diagnosis; they supplement one another.

Breath sounds result from the vibrations set up by the passage of air

able to remember them distinctly. Pathologic conditions are most apt to be indicated in the expiratory phase. Abnormal breath sounds may indicate cavity, consolidation, pleural effusions, etc.

different diagnosis of fluid, tumors or any gross pathological condition. Whisper sounds result from the vibration produced by whispering. These

indication of these conditions than voice sounds, because they do not set up sympathetic vibrations in other parts of the thoracic cage.

Rales are the most common adventitious auscultatory sound. They result from the passage of air through matter is present to set up new

tion as bronchial tracheal and sound are divided ordinarily into two groups, dry and moist. They are quite common to many pathologic chest conditions. Rales high pitched in tone indicate consolidation, a low pitch indicates emphysematous tissue in the area.

Tactile fremitus is the appreciation of voice sounds by the sense of touch. It is the vibration set up by the voice sounds which can be felt through the chest wall. It is most pronounced in the lower part of the chest, and is most easily appreciated in the axilla. It is most pronounced in the lower part of the chest, and is most easily appreciated in the axilla. It is most pronounced in the lower part of the chest, and is most easily appreciated in the axilla.

The analysis and correlation of the findings of a sound study is much easier if a chart is set up. Many such charts have been devised, the one that follows was used in our clinic because it is simpler than most and because one is enabled to interpret the findings in terms of physics.

No scheme of charting is entirely satisfactory, but this one has worked out best for us over a period of time.

For the sake of convenience BS++ and WS+ and no impairment of resonance have been arbitrarily selected as the normal type of breathing (Normal vesicular breathing).

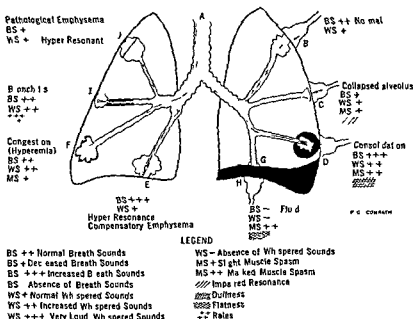


FIG. 2.—The author's chart for the interpretation of physical findings. It illustrates the importance of correlation of the various findings in determining the nature of the underlying condition.

BIBLIOGRAPHY

- SCHULMAN C. Pulmonary mechanism for breath sounds (preliminary report) Quart Bull Sea View Hosp 8:114 1946
SINGER J J

culosis

Maryla

2. LABORATORY METHODS IN DIAGNOSIS

LITTLE need be said about the importance of the laboratory to diagnosis

Sputum—Sputum is primarily useful as a test material for bacteria acid fast bacilli and fungi. The diagnostician should not however neglect gross appearances. Color and odor and amount of sputum should always be noted. In pneumonia for instance large amounts of sputum indicate an extensive area of edema with consequently a rapid spread of the infecting bacteria. A severe infection can be anticipated.

The sputum should be closely studied for even the faintest traces of

the sputum of a patient only upon the sixth third examination. On the other hand many pathogenic bacteria and fungi can be found in healthy adults and they can be presumed to be pathogenic in a particular instance only when they are repeatedly demonstrable in abnormal quantities.

A plain smear does not always give conclusive results in such instances the sputum should be cultured. Such cultures are ordinarily done by laboratories and need not be discussed here. However it may be pointed out that again a single result is not necessarily conclusive. It is particularly difficult to culture tubercle bacilli and several different culture media may have to be tried.

The collection of specimens is sometimes a problem especially with children. An applicator in the pharynx sets up an irritation cough which usually expels some particles of sputum. A laryngeal mirror may also be used smears can be made from the secretions coughed onto the mirror. These methods of irritating the pharynx should be used on all patients whose sputum when collected by the normal method fails to demonstrate tubercle bacilli.

In extreme cases secretion can be secured directly from the bronchial tubes by aspiration through a bronchoscope. A great advantage to this method is that the examiner knows from which lung a specimen is secured. Hence the collection of samples of sputum for various analyses is routine procedure when a patient has to be bronchoscoped for any cause.

by the patient. In 57 cases reported by Clerf and Herbut 47 were diagnosed as carcinoma on the basis of secretion studies where as such diagnosis was possible on the basis of other evidence in only 31 of the cases. This technic when more widely applied should be of inestimable benefit to the control of a disease where early diagnosis is so necessary.



FIG 3—Sputum specimen reveals cells which suggest the presence of malignancy (Papanicolaou stain). Later proved positive by biopsy of the lesion. (Courtesy of S. M. Farber.)

The technic is quite simple. The secretion should be secured if at all possible from the larger bronchi, the most likely area for malignancy. Smears should be made as soon as possible and fixed while still wet with a solution of equal parts of ether and 95 per cent alcohol. The smears should then be stained by the Papanicolaou technic. Carcinoma cells are easily distinguishable (see Fig 3).

Where a patient is suspected of tuberculosis although his sputum is

apparently free of bacilli a stomach wash may sometimes confirm the disease. The technic of stomach washing is as follows. The examination is made preferably before breakfast. The patient is first given a glass of water. A stomach tube taken out of ice water is placed between the patient's teeth and his head is brought forward by a gentle pressure on the back of the head until his chin rests on his chest. He is then asked to breathe through the mouth and at the same time to release the tube from between his teeth. At the moment of release the tube is quickly but gently inserted into the larynx and on into the stomach 3 or 4 inches beyond the mark. The bulb is compressed and released which should withdraw 2 to 3 ounces of liquid. This liquid is centrifuged for forty-five minutes; the sediment can be treated and cultured in the same manner as sputum.

Blood—Information gained from a study of the patient's blood is useful in spite of being for the most part non-specific. In all instances of obscure visceral symptoms blood smears should be examined microscopically for malarial and filarial parasites and for *Spirochaetales* particularly *Borrelia*.

condition in the chest. Single readings of a leukocyte count are not however reliable due to the wide variation that can result from non-specific conditions.

Polycythemia (an excess of red blood cells (up to thirteen million per cubic millimeter)) is frequently associated with pulmonary congestion.

disease (eosinophilic infiltrations of the lung) is frequently characterized by eosinophilia. Again however an increase in eosinophils is characteristic

TABLE I

Method	Men	Women
Westergren	0 to 15 mm	0 to 20 mm
Wintrobe	0 to 6.5 mm	0 to 15 mm
Cutler	0 to 8 mm	0 to 10 mm

An accelerated rate always suggests an organic disease especially an inflammation. It can mean active infectious tuberculosis, localized suppurations, internal hemorrhages; it is a characteristic of malignancy in the later stages.

A normal rate is usually maintained in the case of wasting diseases and in the case of non-malignant tumors unless they are disintegrating.

A below normal rate does not ordinarily occur in diseases of the chest.

The sedimentation rate of erythrocytes is valuable as a clinical guide to the progress of tuberculosis; a falling rate indicates improvement of the condition, a rising rate the progression of the disease. A suddenly rising rate indicates the development of complications.

Red cell fragility, the amount of prothrombin in the plasma, and coagulability and clot retraction time are general characteristics of the blood.

ing progress under treatment. The various techniques involved cannot be here discussed, but detailed discussion is available in various treatises on laboratory diagnosis.

Chemical tests of the blood are also important, since the presence of sugar, abnormal amounts of cholesterol, or an abnormal relationship between phosphorus and calcium suggests certain pathological processes. Laboratory analysis of bone marrow may also be helpful, and new techniques of obtaining specimens makes such analysis feasible.

Urine—The urine analysis is very important in the study of chest conditions, particularly in the detection of amyloidosis. This disease is found in connection with lung suppurations of long standing and tuberculosis; it also occurs, however, in diseases not associated with the chest.

Pulmonary tuberculosis is frequently associated with renal tuberculosis. The latter can be distinguished with some care by urine analysis. Thorough cleaning and irrigation of the parts below the kidney are necessary to eliminate smegma, which are hard to distinguish from tubercle bacilli. Also the test may have to be repeated over a period of time, like a sputum check; one or several negative tubercle checks of urine are not conclusive evidence of absence of infection.

The urine check for tubercle bacilli is most important in suspected cases of primary tuberculosis; the bacilli are often found in urine, seldom in sputum.

Bence-Jones protein in the urine is found in cases of blood and bone diseases, sometimes confused with chest conditions or accompanying them.

Feces—The most important condition to be observed in a feces examination is the presence of occult blood. This may be an indication of an open lung lesion from which blood has oozed into the pharynx and been swallowed, although such an origin cannot be presumed until the possibility of some lesion of the stomach, such as an ulcer, has been ruled out.

A careful study must be made of any fungi and protozoa in the feces, particularly amebæ. The *Endamoeba histolytica* and the *Endamoeba coli* are sometimes associated with symptoms of lung suppuration in cases where liver abscesses rupture into the pleural cavity. This possibility should be considered in suspicious cases of lung pathology.

Effusions—These are of two general types, transudate and exudate. They are usually distinguishable by casual observation, but such observation should be confirmed by laboratory analysis.

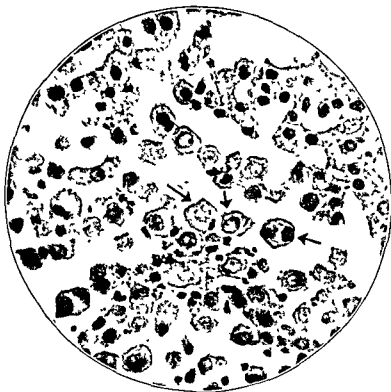


FIG. 4—Microscopic examination of paraffin section of centrifuged sediment of thirty

cells are present

Diagnosis—Carcinomatous extension or metastasis to the pleura

A transudate or serous effusion, is essentially an edema fluid, non-inflammatory in character, resulting from circulatory disturbance. It is clear and ranges from colorless to a light yellow. It is usually alkaline and

has a specific gravity of from 1.006 to 1.018. It has few cellular elements and yields little or no fibrin. It is a symptom of a rather general nature.

Exudates vary widely in color and appearance, but they are in general thicker in consistency and less clear in appearance than transudates. They are rich in cellular elements and fibrin may be present in quantity. Specific gravity is above 1.018. Lymphocytes predominate markedly in a cell count. Blood may be present. The presence of cholesterol crystals and chyle, though rare, is especially significant to diagnosis. Exudates indicate an inflammatory condition of some sort or a tumor. The examiner needs to be aware of any number of possibilities in studying exudates and must care-

method the fluid is decanted and the sediment is poured into a 50 cc. centrifuge tube and centrifuged for at least twenty minutes at a moderate speed.

ened with

Zenker's

treated as ordinary tissue, run through alcohols, embedded in paraffin and stained with hematoxylin-eosin. The tissue is cut from above down so as to include all cellular elements. Carcinoma cells, when present, are easily recognized (*see* figure 4).

Tuberculous exudates somewhat resemble transudates. They are a clear yellow or sometimes a faintly greenish yellow, with an albumen content of about 4 per cent. Acid-fast bacilli can usually be demonstrated. If direct smears fail to reveal such bacilli, cultures and guinea pig inoculations should be made.

BIBLIOGRAPHY

3. MECHANICAL AIDS TO DIAGNOSIS

In addition to the methods of observation and analysis thus far outlined the chest diagnostician has available several techniques for chest examinations involving mechanical aids. These aids range from the simple postural drainage table to the bronchoscope which requires special training for operation. All of these mechanical devices are important to treatment as well as to diagnosis. The more recent ones are aimed at direct visualization of the viscera—an enormous advantage to the diagnostician. As the possibilities of mechanical aids to visualization are further explored and as they are more widely practiced the exploratory operation of former days would become increasingly rare.

Postural Drainage—This procedure consists simply of the patient assuming a lowered head attitude to make easier the expulsion of fluid or pus in the pulmonary cavities and bronchi. It is primarily directed to the relief of discomfort caused by the presence of large amounts of such material. The physician who is familiar with it will find a postural drainage table

The patient to whom the treatment is applicable will ordinarily know the position he must assume for easiest expulsion of the purulent material.

Postural drainage is also of considerable use in diagnosis.

The author has seen many patients in which a fluoroscopic examination after such drainage revealed cavities previously undiscernible. All postural drainage should always precede the use of iodized oils for outlining bronchiectatic cavities.

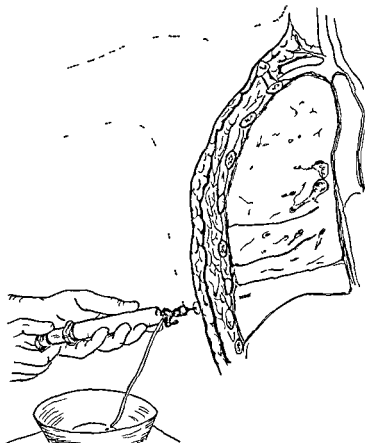
Diagnostic Thoracentesis—A thoracentesis should be undertaken whenever symptoms of acute pressure in the thoracic cage are present. Such pressure is generally manifested by dyspnea, cyanosis (redness) of the lungs. In addition it is useful to diagnosis as a method of obtaining pleural fluid for laboratory analysis and it is a necessary preliminary to diagnostic

tion should be washed with tincture of iodine followed by 70 per cent

injected as the needle is introduced deeper into the tissues until the pleural cavity is reached. Then the piston of the syringe is slowly withdrawn. If

no fluid is obtained from the pleural cavity, the position of the needle is changed both as to depth and direction and another aspiration is done.

Ordinarily aspiration should be repeated until the cavity is emptied of fluid. However, not more than 1000 cc of fluid should be withdrawn at one time unless it is replaced with one half the amount of air. Frequently a lung which has been compressed by the pressure of fluid for a long time may not expand when the fluid is removed. In such a situation the aspiration produces a high negative pressure in the pleural cavity and may cause



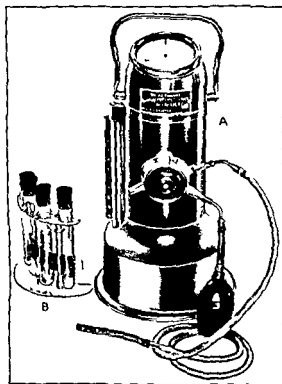
(C. Inc.)

considerable discomfort and even lung rupture if the suction or negative pressure is strong enough to pull a fixed mediastinum toward the affected side. Such suction may also give rise to marked embarrassment of cir-

duction of air

Laboratory analysis of the aspirated fluid should of course be made as outlined in the section on effusions

The operation is not without some danger. Infection of the pleura, air embolism or interstitial emphysema may develop. Very rarely sudden death has resulted. The chest explains the dilator reflex.



turely of
cerandi
(Made

and its usefulness in many situations far outweighs the slight hazards involved

Diagnostic Pneumothorax.—The usefulness of pneumothorax to treatment of pulmonary lesions is well-known, less well-known is its usefulness to diagnosis. Diagnostic pneumothorax is a simple procedure that may be of invaluable aid when ordinary roentgenograms have failed to make pleural conditions clear

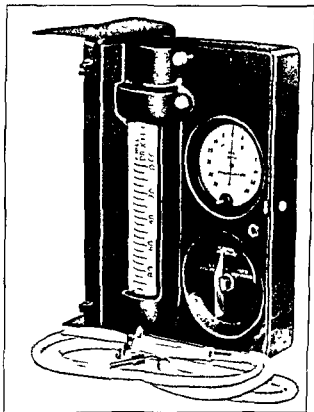


FIG 7—Smaller type pneumothorax apparatus designed by the author with 100 cc capacity. The apparatus can be easily carried in the physician's satchel. (Made by Phillips Drucker Co., St. Louis.)

Several pneumothorax machines are currently available. The author has designed two such machines, both portable, one with a capacity of 1000 cc and a much smaller and cheaper one with a capacity of 100 cc. The larger machine is bi-chambered, with mineral oil that has been pumped into the upper chamber. The machine operates by measuring pressure.

The machine is filled with the amount of air desired and the patient prepared for the operation. The needle is inserted into the pleural cavity

The first step is to obtain a reading of the pleural pressure present in the cavity by turning the commutator valve to 'Pleural Pressure'. If this pressure is not abnormal the commutator valve should be turned to 'Air' which begins the injection of air into the pleural cavity. As much air as desired can be injected with either machine. The pleural pressure should be frequently checked by turning the handle again to 'Pleural Pressure'.

If the patient shows symptoms of distress the air in the pleura can be immediately withdrawn by turning the handle to 'Vacuum'. Both machines offer smooth, easily controlled injection of air combined with a high degree of portability.

Röntgenograms taken with the free space of the pleural cavity filled with air are most helpful in definitely locating an abnormality as in the lung, pleura or mediastinum. Such röntgenograms may also reveal tumors of the pleura and the outer surface of the lung, mediastinal masses and calcification of the pleura that were not evident before the pneumothorax. This technic may also be helpful in distinguishing tumors from fluid. An artificial pneumothorax by partial displacement of the lung has made visible a bronchiectatic atelectatic lobe previously hidden in the cardiac shadow.

A second technic with diagnostic pneumothorax that the author has found helpful is the introduction of air in a pleural cavity containing some fluid. By moving the patient so treated under a fluoroscope the fluid level can be observed. By this means the examiner can often determine whether the pleura is smooth or rough and can locate adhesions or irregularities. The contours of the lung can be mapped out by turning the patient upside down; the diaphragm can be examined.

The introduction of air for diagnostic pneumothorax should be done with the same care as for pneumothorax treatment. Apprehensive patients

diagnostic in

1. There is

kind and it

should not be resorted to unless the information is unavailable by any other method. Air embolism is probably the chief danger to be considered before undertaking any pneumothorax, but the author has rarely seen severe reactions to the procedure.

Bronchoscopy—In the past the bronchoscope proved itself by its contributions to lung surgery; progress in such surgery would have been slower without the instrument. This is particularly so in the case of neoplasms of the tracheo-bronchial tree and tuberculosis. In recent times bronchoscopy has become common and physicians trained in the art are to be found in almost all large communities; the use of the bronchoscope in the diagnosis of pulmonary lesions is routine procedure whenever such inspection of the bronchial tree

and training
and Esophagoscopies) or St
(page 502 and

hesitation whenever failure of conclusive diagnosis without it seems likely. It should always be used, of course, in cases of bronchial obstruction or obstructive atelectasis or emphysema. It can frequently be of help in diagnosis of asthma. Bronchial tumors should always be viewed directly if their location makes it possible and biopsy specimens should be taken. The focus of infections that are not visible on a roentgenogram can sometimes be determined. Bronchoscopy has proved very helpful in the diagnosis of tuberculous lesions in the tracheobronchial tree where other evidence such as acid fast bacilli in the sputum is lacking. It has in the past permitted diagnosis long before other evidence was conclusive.

No chest surgery should be undertaken of course, without as complete a preliminary examination as possible, information gathered bronchoscopically may be vital.

Thoracoscopy—This is actually a surgical procedure and should be so regarded. Although it is primarily useful in severing adhesions between lung and chest wall that prevent artificial pneumothorax, it may be resorted to when pathologic conditions in the pleural cavity otherwise resist diagnosis. It should not be undertaken lightly, the examiner needs to be thoroughly familiar with the structures of the thoracic cage and must exercise extreme care in avoiding the large number of blood vessels and nerves in the chest wall.

The pleural cavity should be thoroughly drained if fluid is present and an artificial pneumothorax large enough to permit the manipulation of instruments should be established. A complete roentgen-ray study should be made before the operation with films taken in multiple positions. Such films should be carefully studied to determine the location of adhesions because if the thoracoscope is inserted above one of them it may easily

The method for thoracoscopy that the author uses is as follows. After a satisfactory pneumothorax is established the patient is prepared with nembutal and morphine as outlined in the section on diagnostic pneumothorax and the skin is anesthetized with a 1 per cent solution of novocain. An incision is made with a small knife in the intercostal space nearest the indicated pathologic condition to be examined. Through this incision the trocar and cannula are introduced into the pleural cavity. With the instrument in the chest the trocar is removed and a thoracoscope is introduced through the cannula. The examiner now can obtain a direct view of the pleural cavity. With experience he will be able to visualize the con

calcification on the pleura, areas of congestion and small abscesses on the lung. Adhesions and tumor masses are of course easily evident. A complete examination will include a specimen of the pleural fluid, if none was secured by thoracentesis and specimens of any suspicious mass for biopsy.

The author has designed a thoracoscope for direct inspection of the pleura

which permits the simultaneous use of several instruments with only one opening in the chest wall. This is possible because of a universal adapter on the proximal end of a small oval-shaped cannula which has two openings—one for a small thoracoscope of the foroblique type and one for the operative instruments. Another method of manipulation of instruments within the chest under direct vision requires a second puncture with another trocar and cannula. The introduction of instruments is of course necessary to the severance of adhesions and is helpful in diagnosis of pathologic conditions.

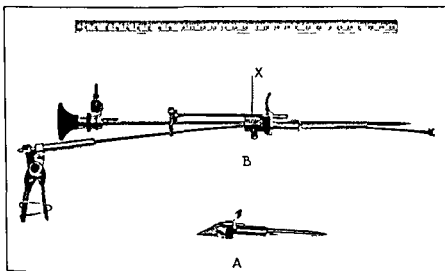


FIG. 8—Photograph of Singer's universal thoracoscope assembled (made by Phillips Drucker Co., St. Louis, Mo.) A shows small oval trocar and cannula, B shows the assembled instrument, X is the universal adapter through which the thoracoscope and operative instruments can be inserted.

BIBLIOGRAPHY

- CAPP, J. A., and LEWIS, D. D. Blood pressure-lowering reflexes from irrigation of the
 13 223 1944
 the Chest. Phila
 in of technique
 10 67 1924

4. ROENTGENOLOGY

In the comparatively short time that has elapsed since Roentgen's observations in 1895 roentgenology has become a formidable technical science the subject of voluminous literature. It is impossible to summarize much less to extend the limits of this science in the space allotted to roentgenology in this book. The best service that can be rendered here is to suggest a perspective to the physician using the roentgen ray and to outline procedure.

Perhaps the chief limitation in the use of the roentgen ray in diagnosis is a mental limitation on the part of diagnosticians. Even in large clinics where it is used extensively there is a tendency to exhaust roentgen ray possibilities with a single anterior posterior view or a single stereoscopic study. Roentgenology is a flexible and supple diagnostic instrument that suffers from such over-simplification and over standardization.

More imaginative use will result from constant awareness of the physics involved in the process. The roentgenogram or the fluoroscopic screen records the relative amounts of a certain kind of light reaching its surface from a cathode tube. Except for its shorter wave length this light is much like ordinary visible light. It diffuses inversely as the square of the distance from the source so that if intensity is taken as 1 at 1 foot it will be $1/9$ at 3 feet and $1/49$ at 7 feet. This light travels in straight lines. Intensity can be varied for best vision at the source the cathode tube. These factors of squared diffusion, linear movement and variable energy are useful to the physician. They must be kept in mind in making and interpreting roentgenograms.

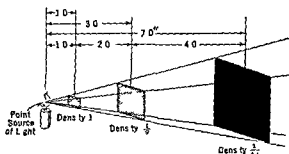


FIG. 9 — The principle of inverse square law

roentgen ray indicates the thickness of structures through which the rays pass

The roentgen ray, then, is a flexible medium

where is, then, no "ideal" - not exact on tissue none
 such a compromise may or may



Fig. 10—A schematic drawing representing the path of X-rays through various media. The lines represent the path of the rays, showing how they are deflected or absorbed by different materials.

not be desirable it is probable in fact, that variation from the typical one way or another would in most instances furnish more information. If delineation of lung markings is desirable best results may be obtained with a minimum of exposure may be used. The relative

at present not clearly defined. It is obvious that the roentgenologic examination will be most efficient if the examiner has at his command the previously ascertained diagnostic information—history, etc. Roentgenologists have suggested that with the simplification of operation of the roentgen-ray machine that is in progress, the roentgenologist will cease to be a specialist at the command of the physician, and the diagnostician and roentgenologist will become one and the same person.

At the present time, the most satisfactory solution is the presence of the diagnostician at the roentgenologic examination. Greater efficiency is obtained if the roentgenologist is previously acquainted with all pertinent information—history, symptoms and physical signs.

Fluoroscopy and roentgenology are complementary to each other, and the former should never be omitted if it can be avoided. Fluoroscopy should of course precede the taking of roentgenograms. It will usually be possible to examine the patient standing before the screen, but if this is not possible a sitting posture is the next choice. A tilting table should be on hand to make a maximum number of angles of view possible.

A standard procedure for the fluoroscopic examination will shorten the time necessary and minimize exposure. The examination should be begun with the patient in the anterior posterior position. The examiner should note

specific areas of the chest concentrating especially on any area suspected of abnormality on the basis of symptoms or physical signs. The apex of

The patient should then be viewed at an angle of about 60 degrees to the fluoroscopic screen with particular attention to the mediastinal structures. After this view is completed from each side the posterior bases of the lungs and posterior diaphragms should be viewed laterally. Routine

examination can be completed with a posterior anterior view noting again the bases, although right and left obliques posteriorly may be included.

Since a rapid examination is important to avoid over-exposure the best procedure is to complete this (or another) fixed routine before a tangential lateral decubitus or other views suggested by an abnormal shadow are attempted. The examiner should however include in the fluoroscopic examination any view his ingenuity suggests might be informative.

The ideal situation obtains with recent equipment by which spot roentgenograms can be made as needed during the fluoroscopic examination. Otherwise roentgenograms, including anterior posterior and lateral views for localization should be ordered as needed. Although stereoscopic examination can aid in localization of abnormalities the ease with which misinformation can be derived from incorrect positioning of the films and other inaccuracy makes it somewhat unreliable and the author shares the growing preference for an anterior posterior view combined with a lateral view for localization when this is possible. It is much simpler to properly position the patient before the screen so that the two views will be at right angles to each other than be certain of stereoscopic localization.

Some care must be used in interpreting roentgenograms. Although they may show detail not visible by means of the fluoroscope because they represent a very short time interval and are not readily re-checkable there is possibility of error. A blood vessel seen longitudinally appears as a most suspicious hard round dot. A film flaw may seem to be an area of emphysema. Superficial blemishes scars or nipples of pendulous breasts are not always easily identified.

Most of the secondary aids to roentgenologic diagnosis are best postponed for a second examination. Some roentgenologists have a barium mixture or barium meal at hand during all examinations to outline the esophagus if necessary and to aid in distinguishing the exact shape and position of the adjacent heart and great vessels. Such a practice is useful. Where the roentgenologic examination follows rather than is concurrent with the fluoroscopy a Visholm grid or Potter-Bucky diaphragm may be added for this part of the examination. This procedure necessitates a kilovoltage and exposure time.

in which the pathologic or dense bronchogenic effusions or deformations are effectively established by simple fluoroscopy and roentgenograms both section roentgenography will frequently yield further information. This technique variously described as tomography, planigraphy and laminagraphy, planigraphy and laminagraphy, is a method of motion to blur out the area in focus throughout the exposure to a single plane within the body. This plane gives much sharpness of detail because detail above and below it is blurred out of the film.

Body section roentgenography is extremely useful in depicting thin-walled cavities hidden by surrounding pulmonary infiltration or in demonstrating motion to blur out the area in focus throughout the exposure to a single plane within the body. This plane gives much sharpness of detail because detail above and below it is blurred out of the film.

strating thick walled cavities or areas of destruction within dense fibrosis or consolidated tissue. It is always of value in the investigation of

be used for exploration.

Examination of the tracheo bronchial tree following the introduction of radio-opaque iodized oil is sometimes of great value in making a diagnosis particularly in chronic diseases affecting the lungs and bronchi.



Fig. 1. Method of injecting iodized oil (I. J. Singer). A. Method of

Before bronchography is performed the patient should be tested for sensitivity to the particular iodized oil being used. It is also essential that the patient also be checked as preparing the patient for b the mucous membranes of cough reflex and facilitating the production of more satisfactory roentgenograms. Patients should be carefully questioned as to cocaine sensitivity since fatal collapse may occur in a sensitive patient. Iodine sensitivity is

quite frequent but the reactions are not quite so severe usually being manifested by rhinitis and conjunctivitis together with moderate caria. If a patient exhibits a sensitization to iodine, it is better to perform the procedure with modern methods.

cor

iodi

T

bronchiaphy all being administered the oil is
grains obtain
or brominized

1 Supraglottic

ed through a straight tube which is placed at the level of the tongue and pulled out deeply and not too far, having the patient assume the position of the head. Practically all methods of injection with the exception of the aspiration method call for the use of a local anesthetic which is rarely necessary when this method is employed. This method is usually not satisfactory for children but it is effective in about 85 per cent of adults.

b Supraglottic Method — The oil is introduced into the hypopharynx through a curved cannula under local anesthesia. By breathing the patient is able to aspirate the oil into the trachea and bronchi.

c Passive Technique (Ochsner) — The mouth is closed and the patient is able to aspirate the oil into the trachea and bronchi. The mouth is closed with a 10 per cent solution of novocaine. The anterior pillar is given 3 or 4 cc. of a 3 per cent cocaine solution and protrude the tongue toward the affected side and breathe. The patient is placed behind a fluoroscopic screen and just previous to the injection the anterior pillars are again painted with 10 per cent cocaine. While under the screen the patient is instructed to take 10 cc. of the oil into his mouth and to aspirate it in the same way as he is aspirated the novocaine.

2 Transglottic — a Catheter Method — Under local anesthesia with cocaine and novocaine a catheter is introduced into the larynx and trachea during bronchoscopy or employing a laryngeal mirror after inserting the catheter through the nostril. When the catheter is in place as seen under the fluoroscopic screen the iodized oil is injected by means of an attached syringe and directed into the desired bronchi.

b Intubation Tube — This method is essentially the same as that described above excepting the use of an intubation tube with a small opening for breathing and the other for injection.

3 Transcutaneous method — The oil is injected with a needle or a trocar to which is attached a curved cannula. The trocar is drawn and a small amount of novocaine is injected. The iodized oil is then injected.

It is usual

tion of iodized oil but occasionally an entire lung may be completely mapped with iodized oil at a single examination. Frequently examination of more than one lung may be undertaken but this procedure is more time consuming and more difficult on the patient as well as more difficult to interpret roentgenographically because of the overlapping of the bronchi as seen in the oblique and lateral projections.

Immediately following the procedure the patient is placed in the position favoring drainage and coughing is encouraged to aid in expelling as much oil as possible to avoid filling the alveoli with the contrast media.

During recent years a new method of chest roentgenography making possible the use of survey methods to check large segments of the population has been developed. By the use of photofluorography employing film on rolls cheap and efficient surveys can be made of large groups of people. The size of such roentgenograms taken singly or stereoscopically varies from 35 mm to 4×5 in the larger being preferable though more expensive. Although this technic was developed for tuberculosis control asymptomatic malignancy silicosis and other abnormalities are frequently first noted by this means. The small but persistent percentage of pathologic conditions thus uncovered thoroughly justifies the technic however individuals thus examined should be warned that they cannot ignore chest symptoms because findings have been negative.

Apparatus especially designed for chest survey examinations are available in various types and capacities and vary from attachments applicable to ordinary standard roentgenographic equipment to mobile bus and trailer units housing every conceivable unit necessary for the production of the survey films. Many of these mobile units contain their own power plants

essentially
are fluorescent
s of a photo-

graphic camera employing a very high speed lens.

While photofluorographic methods have reached a high degree of accuracy it is felt that they should be employed only in survey of large groups for the detection of pathologic conditions and that actual diagnosis of disease done by further employing fluoroscopy and standard roentgenographic examination with 14×17 in film in all necessary projections. During the past few years much improvement in actual mechanics of photofluorographic methods has been described including the Morgan phototimer and various automatic devices containing motor driven cameras with which the film is advanced on the roll automatically between exposures without the aid of the technician.

As with other diagnostic techniques the detection of an abnormality depends upon thorough familiarity with normal appearances and permissible variations from the normal. Anatomy remains basic to medicine. Such anatomy as seems appropriate to this book has been consigned to later sections for the most part. However it needs be emphasized here the vital necessity of complete command by the diagnostician of the lobes of each lung. Probably the most frequent error in the diagnostic use of the roentgen ray is the confusion of lobes or lobules which are atelectatic or consolidated due to a pathologic condition in their bronchi for a primary

abnormality. Tumors are particularly apt to be thought much larger than they really are. This diagnostic error is most easily avoided if the normal location of such lobes is understood.

BIBLIOGRAPHY

- 1936
 ANDRE R. J. R. and STAVA R. J. Planigraphy: mathematical analysis of methods describing of apparatus and experimental proof. *Am J Roentgenol* 38: 145, 1937.
 ARCH BALD F. C. A ray demonstration of pulmonary changes in tuberculosis by lipodol
 ———— Technique of bronchoscopic pneumography with lipodol. *Surg. Gynec. & Obst.* 47: 673, 1928.
 BALDWIN D. F. P. A. C. S. I. C. F. P. B. S. B.
 CARVALHO I. de and MONZ L. The visibility of the pulmonary vessels (angiopneumography). *Acta Radiol.* 14: 433, 1933.
 COLE A. J., MOORE S. and DEAN L. W. Relationship of paranasal sinus disease to ocular disorders: the roentgen method of investigation by laminagraphy. *Laryngoscope* 49: 374, 1939.
 DUBOIS K. Interpretation of best roentgenograms. *J. A. M. A.* 101: 1807, 1933.
 ———— Comparison of roentgen ray densities with pulmonary pathology. *Am J Roentgenol* 32: 317, 1934.
 ———— Technique and interpretation of chest radiogram. *Tubercle* 17: 406, 1936.
 EXNER F. B. and RIGLER I. G. Relative value of stereoscopic and single films in
 JACKSON C. P. Bronchoscopy and esophagography: brief consideration of technique. *Surg. Gynec. & Obst.* 44: 790, 1927.
 KIEFFER J. Laminagraph and its various applications and applications of planigraphic principle. *Am J Roentgenol* 39: 437, 1938.
 KUGLE D. S. Right middle lobe and lateral roentgenogram. *J. Thoracic Surg.* 6: 66, 1937.
 LIAN C. DUBOIS and NAVARRE P. Nonpenetration into the trachea of certain supposed intratracheal conditions. *Bull. et m. m. Soc. m. d. h. p. d. Paris* 46: 470, 1932.
 LYNA H. L. Lung mapping by injection of indium mixtures. *New York M. J.* 114: 82, 1921.
 MAIER H. C. Roentgen aspects of upper retro-esophageal pulmonary disorders. *Am J Roentgenol* 43: 165, 1940.
 MOORE S. Body section roentgenography with laminagraph. *Am J Roentgenol* 39: 514, 1938.
 ———— Body-section radiography with laminagraph in pulmonary disease. *Am Rev. Tuberc.* 38: 538, 1939.
 ———— Body-section radiography. *Radiology* 33: 600, 1939.
 ———— Practical applications of body-section roentgenography. *Am J Roentgenol* 44: 24, 1940.
 ———— Body-section radiography in malignancy of lower respiratory tract. *Surgery* 8: 924, 1940.

THE THORACIC CAGE

5. DEFORMITIES, FRACTURES AND INFLAMMATIONS OF THE THORACIC CAGE

portant anomaly

There are however a rather large number of abnormalities and disease processes of interest and importance to the chest physician that do not merit individual treatment. These are congenital and post fetal deformities, fractures and subcutaneous lesions.

Deformities—Any pronounced abnormality of the thoracic cage will be mirrored in a displacement or distortion of the organs within that cage. Such displacement or distortion can be quite severe without causing a serious impairment of function. On the other hand progressive dysfunction is common to many of the deformities and some of them can produce in extreme instances pronounced clinical symptoms.

Funnel chest the peculiar funnel shaped depression of the sternum can be congenital or traumatic in origin. In some instances it seems to be hereditary. Only rarely does it produce symptoms of itself although it may seriously complicate other thoracic conditions. The depression usually deepens in the course of time and vital capacity may be diminished to a

chest it may distort and displace the thoracic and upper abdominal viscera to a degree sufficient to seriously impair function.

The *barrel-shaped thorax* is defined by an abnormal anteroposterior diameter. It is a post fetal developmental defect and can be due either to obstructive emphysema of the lungs or to postural emphysema. The latter condition arises from a disease of some nature that deforms the spine; the resulting barrel shaped thorax in this rarer condition no indication of impaired function. When it is due to obstructive emphysema however it is usually accompanied by marked impairment of visceral function. Dif-

monocardiac failure so marked in individuals with skeletal deformities may well be due to the accompanying thoracic distortion.

Fractures of the Thoracic Cage—Traumatic fractures of the thoracic cage are of course common with the ribs being primarily involved. Diagnosis can usually be made by inspection and palpation although a large hemotoma may exaggerate the extent of the injury. Roentgenograms of simple fracture of the bone oftentimes fail to show the injury and of course are of no help at all in the rare cases encountered of cartilage fracture.



FIG 12 A forty eight
 anterior chest con-
 stant Roentgenologic
 birth with depression
 of the chest wall and
 displacement of the
 heart posteriorly and
 to the left. Diagnosis
 Pigeon breast.

Of special note are the fractures of normal bones through sudden straining or twisting of the thorax. These are not rarely encountered in diseases of the lung particularly tuberculosis where violent paroxysms of coughing are common. The appearance of a new area of pain in a patient under observation might easily confuse the examiner in such an instance.

Pathologic fractures are defined as those which arise in the bones because of a previous weakening due to a disease process—the malignant growths

diagnostician since the origin and primary site of the disease is usually the ribs. A bone so affected will show round or oval punched out areas in a roentgenogram. Only rarely will such a single tumor focus be found—ordinarily they will be numerous. Bence-Jones protein is found in the urine in cases of multiple myeloma. The origin of the disease is obscure but it seems to be a general condition of the skeletal system rather than a specific infection and its typical erosion of the bone may be widespread.



Fig. 9-46. Multiple myeloma.

Diagnosis. Scoliosis of the spine and compensatory emphysema of the lung.

Hyperparathyroidism, or the excessive functioning of the parathyroid glands, may be due to hyperplasia of the glands or to adenomas of the glands. Softening of the bones accompanied by giant-cell tumors and



FIG. 14—Normal thoracic spine (left) and thoracic spine with kyphosis (right).

Diagnosis: Kyphosis

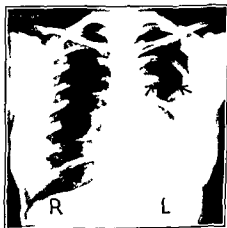


FIG. 15—Surgical fracture of the ribs

parathyroidism. Where it is encountered the irregular densities of the softened bone, the tumors and multiple cysts are usually unmistakable in a roentgenogram. A urine analysis that reveals an abnormal calcium and phosphorus content and a blood test that also shows a high calcium content will confirm the diagnosis.

Paget's disease almost always attacks the skull and long bones first. Its symptom as it appears on the roentgenogram is the regular outline of the bone being replaced by an irregular, thicker and less dense shadow. In such an instance a roentgenographic study of other parts of the skeleton will indicate a similar process going on.

Only rarely does *tuberculous osteomyelitis* lead to pathologic fracture of the ribs; it usually occurs then at the costochondral junction. Unless this condition is demonstrable by roentgen ray it may easily be confused with slipping ribs. Both conditions produce the same local clinical symptoms.

Any cartilage and bone of the extrapleural region of the thoracic cage offer extraordinary difficulty oftentimes to the diagnostician. The deeper infections are usually the most serious, but even the pyogenic lesions of the soft tissues.

Such superficial lesions may have their source in the pleura or may even extend into the pleural cavity if they have been neglected. If several abscesses occur they may be found to be connected. In cases where the abscess aspiration does not reveal an infectious microorganism the possibility of tuberculous origin should be considered.

graphical study made to determine the extent of the abscess cavity. Such superficial lesions may have their source in the pleura or may even extend into the pleural cavity if they have been neglected. If several abscesses occur they may be found to be connected. In cases where the abscess aspiration does not reveal an infectious microorganism the possibility of tuberculous origin should be considered.

sternum, scapula and clavicle.

Pyogenic osteomyelitis of the ribs or sternum is a serious infection. Etiologically the lesion seems a product of the bacteria invading the blood and then, perhaps because of a slight trauma, collecting at a fixation point in the thoracic area. The only evidence available may be pain and associated swelling of the soft tissue. Unless the lesion has advanced to a sufficient degree for ossification to make cartilage deformity visible, it is hard

to detect roentgenographically. Since pyogenic osteomyelitis may so often lead to serious complications the diagnostician is justified in performing an exploratory operation if it is suspected.

Chondritis and perichondritis of the costal cartilages are rarely occurring inflammatory disorders. Occasionally they may be of pyogenic origin or due to rheumatic disease. Usually they are caused by an invasion of the costal cartilage by typhoid bacilli. These bacilli are notorious for their predilection to inactive sequestration within the body after an attack of typhoid fever. Chondritis and perichondritis can result from an infection contracted years before. Whenever an unidentified lesion in the thoracic cage is found in a patient with a history

perichondritis should be considered.

Satisfactory picture of this condition

ally is an abscess in a costal area not accompanied by lesions in nearby bone. An aspiration in such an instance will usually only produce blood.

Syphilitic lesions of the ribs, sternum and clavicle simulate the tuberculous inflammations very closely. The patient's history may point to the cause.

These

exist

pronounced than in a tuberculous infection and an irregular thickening may be observed.

Mycotic infections may invade the soft tissues, the cartilage or bone. Such an infection can be proven by laboratory methods; the specific fungus must be demonstrated. The roentgenogram will not be of great help in delineating the areas of granulation and sinus formation, but it may reveal lesions of the bones in systemic mycosis. When actinomycosis, the most common of the mycotic infections, is discovered, the examiner should suspect a primary lung infection. Actinomycotic inflammations of the chest wall are usually secondary to an infection of the lungs.

BIBLIOGRAPHY

- ALDER, D. D., C. C. B. *Bull. T. Rad.* 17: 323, 1944.
 BALI
 BETT
 BIG

6. THE CERVICAL RIB

Cervical rib is the most important anomaly of the thoracic cage in differential diagnosis. It is present in about 1 per cent of the population but it causes symptoms in probably less than half that number. The statistics reported by different clinics show a wide variation. Symptoms due to cervical ribs are much more commonly found in women than in men.

Etiology—Cervical ribs originate as congenital bands of fibrous tissue which contain osteogenic cells. In some instances such bands develop no further and remain fibrous into adult life. true cervical ribs however result from the gradual ossification of the fibrous band.

Pathology—The cervical rib when found as a cause of symptoms is almost always similar in composition to the bony tissue of other ribs. It may be attached to any vertebrae from the first to the seventh but in an overwhelming number of cases it is attached to the seventh. It is usually unilateral although the presenting symptoms are almost always unilateral and usually found on the right side. It varies widely in development from being merely an extension of the costal process of the vertebra to being a rudimentary first rib joined by cartilage to the sternum. In many cases perhaps in most it is attached by irregular fusion or by a fibrous band to the first rib.

Its importance derives from its relationship to other structures of the area. The very important brachial plexus usually passes directly over it. The subclavian artery arches over it sometimes so acutely angulated as to lead to a thrombosis. The scalenus anticus muscle lies over the cervical rib and is usually attached to it and the subclavian vein and artery are intimately related to this muscle. Also in the area are two branches of the thyroid axis arteries and the suprascapular and transverse cervical arteries. That an anomaly in such an area produces symptoms of severity in many cases is not nearly so strange as the fact that in so many cases it does not.

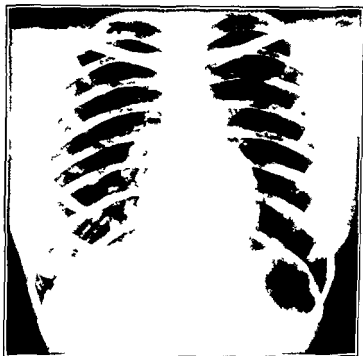
Clinical Symptoms—In many cases cervical rib will only be indicated by pain when lifting a heavy weight or otherwise straining the thoracic cage. Many such cases probably never come to the attention of a physician.

In severe cases the most distressing symptom is pain. It is neuralgic in character beginning in the neck and spreading downward along the arm to the hand. This is due to traction on branches of the brachial plexus. Occasionally it will radiate upward into the head or down into the chest. Paralysis of the muscles of the shoulder arm and hand is frequent in marked instances of this abnormality and rarely Horner's syndrome will be seen.

Physical Signs—In many cases the rib may be palpable. Impaired pulsation may be noted in one radial artery a comparison of the radial artery on one side with the other will usually reveal some difference in pulsation. Areas of paresthesia or anesthesia may be present.

An important diagnostic sign is the production of typical brachial plexus pain when the head is rotated to the opposite side and tension is thus produced on the nerve bundles.

Roentgen ray Findings — A true cervical rib is almost always visible in a roentgenogram. However the examiner may not on the strength of such evidence alone conclude that the rib is responsible for the symptoms since probably a majority of cervical ribs do not produce symptoms. Neither may he dismiss the possibility of cervical ribs if they are not visible in a roentgenogram since the fibrous band that is closely related to the cervical rib but non-opaque may cause the same symptomology.



Differential Diagnosis — A cervical rib may be confused with any other condition that might produce similar pressures on the nerves and vessels of the upper thoracic cavity. The abnormalities most likely to do so are apical lung tumors, bursitis and sarcoma of the humerus or clavicle.

Apical Lung Tumors (Pancoast superior sulcus tumor) — These tumors usually present the physical signs of dullness and abnormal breath sounds. The clinical symptoms of such tumors will often include symptoms of the cervical rib but will usually show in addition cough, loss of weight and strength and bloody sputum. Definitive diagnosis is possible on the basis of roentgen ray examination since such tumors are visible as densities in the upper lung area.

Bursitis — This condition is somewhat more difficult to differentiate

The pain caused in bursitis is often referred down to the arm and is then very much like the pain of a cervical rib. Like the cervical rib other clinical symptoms may be entirely lacking in bursitis. Occasionally an enlarged bursa may be palpated. The roentgen ray offers the surest possibility of distinction again although the roentgenographic picture of bursitis is very often vague and confused.

Sarcoma of the Humerus and Clavicle — In addition to the clinical symptoms of loss of weight and severity of pain which almost invariably accompanies a sarcoma it has a definite roentgenographic appearance and can be distinguished from a cervical rib.

BIBLIOGRAPHY

- ADSON, A. W. Surgical treatment for symptoms produced by cervical ribs and the scalenus anticus muscle. *Surg. Gynec. & Obst.* 85: 687, 1947.
 ADSON, A. W. and COFFEY, J. R. Cervical rib: method of anterior approach for relief of symptoms by division of scalenus anticus. *Ann. Surg.* 85: 839, 1927.
 GRAHAM, E. A., SYNGER, J. J. and BAILEY, H. C. *Surgical Diseases of the Chest*. Philadelphia: Lea & Febiger, 1935, p. 87.
 OLJENICK, I. Bilateral cervical rib: clinical and experimental observations on case. *Arch. Surg.* 18: 1984, 1929.
 WILDER, J. Brachialgia with cervical ribs. *Wien. Med. Wochenschr.* 77: 870, 1927.

7. TUMORS OF THE CHEST WALL

PRIMARY tumors of the chest wall are rather infrequent although at one time or another almost every known histologic type has been reported. Of the benign growths the chondromas are perhaps the most frequently encountered. Angiomas are also found with some frequency. Neurofibromatosis is very apt to present pedunculated neurofibromas on the chest wall.

The malignant neoplasms that originate in the chest wall are usually sarcomatous with chondrosarcoma the most common. Lymphosarcoma is usually a generalized condition but it frequently presents important chest wall manifestations. Carcinoma of the breast is not under consideration here although it may at times involve a large portion of the thoracic cage by extension. Otherwise carcinoma of the chest wall is very rarely primary although it is rather commonly encountered as a metastasis.

Etiology—Benign tumors comprise about one-third of the space-occupying growths in the thoracic cage. For the most part they are thought to originate in embryonic sequestrations of primitive tissue and are thus congenital. This is believed to be true even when there is no gross evidence of such tissue until late in life.

The chondroma group is the exception to this presumption, their typical growth being from cartilage on the surface of the chest wall. They are the most common malignancy not of benign origin.

Pathology—The benign tumors originating in embryonic tissue normally enlarge very slowly until full growth of the host is achieved, then they become stationary in most instances. All of them, however, are capable of resurgence with very rapid growth and they are always potentially malignant. The nevi, pilosi or hairy moles and the cavernous angiomas are particularly subject to malignant degeneration. Irritation is often a demonstrable factor in such degeneration.

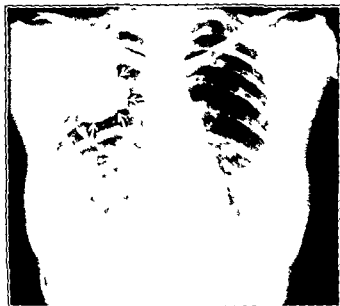
The chondromata are difficult to classify pathologically. Growth is slow but ordinarily persistent. Recurrence after surgical extirpation is not unusual and apparently benign chondromas sometimes produce metastasis. The degree of malignancy is sometimes impossible to determine. In the author's experience benign chondromas may suddenly and highly degenerate into malignant growths.

The chondrosarcoma of the thoracic cage may be of the pleura or soft parts of the thoracic cage is an early occurrence. In many instances the metastatic growth is the first to come to the physician's attention.

Clinical Symptoms—Many benign tumors never present clinical symptoms. Only when the growth achieves unusual size and projects inward will

dyspnea and pressure symptoms occur. Pain is encountered when a tumor presses on a nerve. The chondromas are most apt to achieve a size sufficient to cause symptoms but these tumors are usually visible externally before such symptoms develop.

involvement



normal except for a slight increase in the basal markings.
 Diagnosis: Osteochondromyxosarcoma of the rib.

Physical Signs. Most tumors of the chest wall project out and are obvious to casual inspection. Palpation may elicit the exact location of the tumor with relation to nearby bone and thus suggest its nature but care should be exercised in palpating any suspicious mass since manipulation or other disturbance may increase the possibility of metastasis if the mass is malignant.

A highly malignant sarcoma may pulsate and be warm to the touch. Subcutaneous hemangiomas may first be suggested by local tenderness and a bluish sheen on the skin surface.

In rare instances the major growth of a tumor takes place below the rib cage. If such a tumor impinges upon the lung, its presence will be suggested by abnormal auscultatory and percussive findings.



FIG. 1. Roentgen-ray of chest showing metastatic carcinoma of the lungs and ribs.

on left

Diagnosis. Metastatic carcinoma of the lungs and ribs

Roentgen-ray Findings—It must again be emphasized that a roentgenologic study of any chest abnormality cannot be limited to the dorso-ventral position. With a thorough examination, however, the location and extent of involvement can be determined with a reasonable degree of accuracy. Erosion of bone near a doubtful mass may indicate its location. A growth primary of the lungs or mediastinum will usually distort ribs impeding its cutaneous extension. In some instances a diagnostic pneumothorax preceding the roentgen-ray examination may be necessary to determine with certainty whether or not a tumor shadow appearing in the chest

wall represents in reality an extension of a deeper growth or a growth of the chest wall itself.

A roentgen ray examination will frequently not only definitely localize a tumor but will suggest its nature. New cast round homogeneous shadows suggesting a metastatic growth. The neurofibroma of Recklinghausen's disease is normally situated within the intercostal space and will be suggested by a round clearly-outlined shadow surrounded by notched and eroded ribs due to pressure. Confirmation of the diagnosis of Recklinghausen's disease may be made by the roentgenologic demonstration of

density but quite distinct will be seen at the site of the lesion. On the other hand metastatic carcinoma when it invades the thoracic cage will present a fuzzy indistinctly outlined mass with irregular erosion and thickening of the ribs.

A thoracentesis is of course indicated when fluid is present in the pleural cavity to obscure roentgenologic findings and if a diagnostic pneumothorax is established for roentgenologic purposes a thoraco copy is usually worth while because it furnishes direct inspection of any suspicious mass as well as permits the removal of tissue for biopsy.

Laboratory Tests—A biopsy is the best means of positively identifying a tumor.

against
may be

tumor such a specimen may easily be mistaken for tumor tissue upon gross inspection. The author has encountered several instances in which repeated biopsies from growths which were considered carcinomas on clinical grounds did not reveal malignant cells but in which autopsies nevertheless confirmed the suspected nature of the disease.

Occasionally a punch biopsy or even an exploratory operation may be necessary to confirm a diagnosis. Where a pleural effusion is found a study of the effusion according to the Mindelbaum method may be helpful.

The incidence of tumors of the chest wall does not justify a discussion of the complicated laboratory methods of identifying tumor tissue the material under laboratory analysis as well as other tumor chapters may be of some interest. Numerous treatises such as Bell's *Pathology* and Fung's *Neoplastic Disease* are available that cover the subject extensively.

Differential Diagnosis We have tried in the foregoing sections to

scapular abscesses and Paget's disease of the bone. Cutaneous lesions may sometimes appear to be tumors of the thorax. Impetigo necrositatus may suggest a new growth.

Hydatid Cysts of the Pleura—The hydatid cyst is a rare finding in the pleura when it is encountered there it is usually situated above the right subphrenic space. It can become so large as to simulate the pressure symp-

toms of a neoplasm the rapidity of growth may suggest malignancy. A history of residence in Australia, Italy or South America is suggestive. A biopsy should not be undertaken of a growth in this area if the hydatid cyst is a possibility. Anaphylactic shock usually follows penetration of the thin cystic wall. The possibility of the cyst should be eliminated by the intradermal Casoni test before other diagnoses are considered.

Lung Hernia—This rare condition is easily mistaken for tumor especially for a soft lipoma in a superficial examination. Occasionally a large

reveal air containing lung immediately beneath the skin.

Subscapular Abscess—This condition is likewise rare. It is very often mistaken for a tumor especially a tumor of the scapula. It may become quite large and seem to be attached to the scapula. A needle aspiration however will demonstrate pus. This will definitely eliminate the possibility of tumor and after iodized oil and air injections into the abscess cavity the lesion will usually be seen to be quite distinct from the scapula itself.

Paget's Disease of the Bone—This disease also called *osteitis deformans* is one of the most common chronic diseases of the skeletal system. Although it most commonly manifests itself in the skull and long bones it occasionally is first noted in the bony structure of the thoracic cavity. Metastatic carcinoma may be suspected particularly since Paget's disease

cases roentgenograms of the skull and long bones will show the process there also. A familial history is important since there is definitely an hereditary tendency. Blood chemistry studies will reveal an increased

discussed elsewhere in detail. Roentgenograms will demonstrate that the infection is of a superficial nature and does not involve the thorax and if an empyema ruptures into the soft tissues of the thorax. A roentgen

BIBLIOGRAPHY

- BELL, F T T -
 BROW
 R
 DAUN -
 E
- EA
 FR
 GR
- HERBERT W P Ewing's tumor of rib report of two cases J Thoracic Surg 5 180
 1935
- LAMBERT R A
 LOEN I
 PANCOAST
 tumors
 SAMSON P (1020 1935
 SIMMONS C C Malonant
 Gynee
 SURE H
 bone R
 SUGARBAKER
 biopsy
 SASSMAN M L S
 contrast vasis
 46 74 1941
 SASSMAN M L S
 contrast angiocard ography
 WARREN S and Sommer G N
 to recurrence and metastases
- gment
 cases of the Chest
 J Thoracic Surg 5 180
 J A M A 105
 b
 ,

toms of a neoplasm the rapidity of growth may suggest malignancy. A history of residence in Australia, Italy or South America is suggestive. A biopsy should not be undertaken of a growth in this area if the hydatid cyst is a possibility. anaphylactic shock usually follows penetration of the thin cystic wall. The possibility of the cyst should be eliminated by the intradermal Casoni test before other diagnoses are considered.

Lung Hernia—This rare condition is easily mistaken for tumor especially for a soft lipoma in a superficial examination. Occasionally a large part of the lung may protrude usually it is just a lobe or an edge of a lobe. It may occur through any intercostal space above the diaphragm below the clavicle or even in the neck region. Auscultation will usually reveal breath sounds within the soft sponge-like mass and roentgenography will reveal air-containing lung immediately beneath the skin.

Subscapular Abscess—This condition is likewise rare. It is very often mistaken for a tumor especially a tumor of the scapula. It may become quite large and seem to be attached to the scapula. A needle aspiration however will demonstrate pus. This will definitely eliminate the possibility of tumor and after iodized oil and air injections into the abscess cavity the lesion will usually be seen to be quite distinct from the scapula itself.

Paget's Disease of the Bone—This disease also called osteitis deformans is one of the most common chronic diseases of the skeletal system. Although it most commonly manifests itself in the skull and long bones it occasionally is first noted in the bony structure of the thoracic cavity. Metastatic carcinoma may be suspected particularly since Paget's disease

process there also. A familial history is important since there is definitely an hereditary tendency. Blood chemistry studies will reveal an increased

tation will reveal characteristic sounds of coursing blood within the mass.

discussed elsewhere in detail. Roentgenograms will demonstrate that the infection is of a superficial nature and does not involve the thorax and microscopic examination of the secretions from the sinuses especially if they are tuberculous or fungus in origin will reveal their true nature readily.

Empyema necessitatus—This condition can be mistaken for a tumor if an empyema ruptures into the soft tissues of the thorax. A roentgen

levels produced by the injection of the air

DISEASES OF THE PLEURA .

8. ANATOMY AND MISCELLANEOUS DISEASES

Anatomy The human being has actually two pleura each surrounding a lung and separate from the other. Each pleura consists of two serous membranes the inner visceral and the outer parietal. Normally these two layers are independent of each other and because of the fluid they exude they slide freely one on the other aiding free lung function. Adhesions are very common however and except in extreme cases do not seriously inhibit lung function. The visceral pleura has the chief function of making the lungs airtight the parietal pleura is protective. Both membranes in a healthy individual are resilient and flexible and tough.

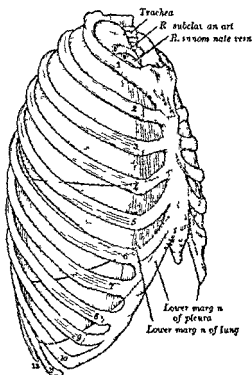


FIG. 19.—Lat. view of thorax showing the relation of the pleura and lung to the chest wall. The right lung is light grey, the left lung is dark grey. (After Gray's Anatomy.)

lungs at
Rare-
due to

congenital weakness but a fetus with any extensive pleural deformity will be still born

The pleura is seldom the site of a primary disease on the other hand because of its intimate relation to the lungs and visceral lymph channels it is often a site of secondary infection In most instances this secondary infection is manifested by the abnormal accumulation of a liquid within the pleural cavity

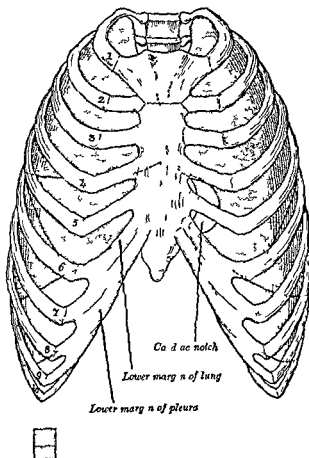


FIG. 20 Front view of thorax showing the relations of the pleura and lungs to the chest wall. Pleura in light grey; lungs in dark grey. (After Gray's Anatomy.)

The pleura may also be the site of tumors which except in rare instances are metastatic and fungus diseases which are generally extensions of primary lung infections. A few conditions of the pleura that represent aftermaths of infections also need to be mentioned.

Calcification of the Pleura — Calcification of the pleura is rarely reported in general practice, but it is not an unusual finding in chest clinics. The reason for such calcification is not understood although it seems to represent a protective mechanism of the body. It does not seem to be a product in any way of a high calcium content in the blood. It occurs rather frequently following a traumatic hemothorax and it is sometimes



FIG. 91. Bullet in right lung and calcified pleura.

at the extreme right apex.

Diagnosis: Bullet in left lung and calcified pleura.

a sequela to chronic tuberculosis or to a chronic empyema. It ordinarily occurs in patches, but the author has seen one patient with an entire pleura calcified. A lung abscess may develop

Because a development of this nature interferes with the function of the pleura and consequently the function of the pleura may prove to be quite serious in it sharp-edged plaques break loose they may damage the lung or produce a spontaneous pneumothorax

to be asymptomatic with this technique ears is to the calcified cold abscess of Pott's disease and distinction can be made between the two in almost all cases by roentgenograms from different angles due to the thinness of the calcified plaques

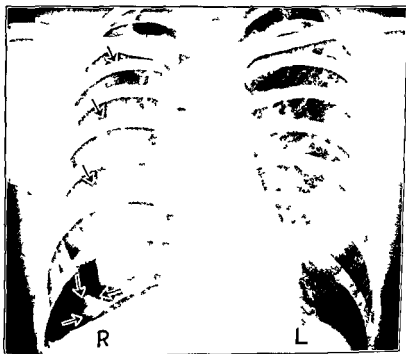


FIGURE 1. Pleural fibrin body (Chest X-ray)

Fibrin Bodies in the Pleural Cavity The mechanism of the formation of fibrin bodies in the pleural cavity is not better understood than the mechanism of calcification. Because they are most often found in a tuberculous patient being treated with artificial pneumothorax it has been

suggested that fibrin bodies are formed from blood arising because of the puncture of an intercostal blood vessel during the pneumothorax treatment
 a torn lesion with

though they are yellowish in color and resemble tuberculous material. They most often appear as irregular dense shadows on a roentgenogram which shift with a change of position. They are in many cases re-absorbed when pneumothorax is discontinued. They are *primarily important* because they are so easily confused with tumors of the lung or chest wall with encysted empyema, echinococcus cysts, an atelectatic lobe of the lung, a pendulous fatty pericardium or calcification of the pleura. A roentgenologic study will almost always indicate the nature of a fibrin body; in rare cases a thoracoscopy might be indicated.

Tumors of the Pleura—Primary tumors of the pleura are rare. Occasionally one finds small benign growths such as fibromas, lipomas, angiomas or chondromas in the subserous layers of the visceral pleura. Such growths do not ordinarily achieve a size sufficient to require surgical treatment.

The chief primary sarcoma. Because of relation in the pleura causes death by interference with the cardiac function. It seldom metastasizes.

Metastatic tumors are much more common in the pleura than are primary tumors. The most frequently found malignant growth is the endothelioma but carcinomas may extend from the lung.

Diagnosis of any tumor of course depends on examination of biopsy specimens. As outlined in other sections although a roentgen ray study of a tumor will often strongly suggest its nature. Malignant growths especially are apt to be accompanied by a pleural fluid and in somewhat more than half the cases of such malignant fluid producing growths a persistent examination of the fluid will reveal the nature of the tumor.

Thoracoscopy when malignancy is suspected is often the best means of securing specimens for biopsy.

In many instances the most serious problem presented by a tumor will be the determination of its exact location. Such determination is usually possible with a multi position roentgenographic study but in some cases further techniques are necessary. Iodized oil in the bronchial tree will frequently reveal bronchial obstruction and so locate a tumor of the lung. Artificial pneumothorax followed by roentgenograms will usually make possible differentiation between a growth in the lung and a growth in the pleura.

Fungus Diseases Fungus diseases of the pleura almost always represent extensions of such diseases from the lungs or skin and so these diseases will be discussed more thoroughly in relation to their appearance in the lungs.

A fungus disease in the pleura most often manifests itself in numerous small sinuses which frequently penetrate the overlying muscles and con-

9. ACUTE FIBRINOUS PLEURISY

FIBRINOUS pleurisy is the most common abnormality of the pleura. It is usually quite benign and a great many autopsies of adults reveal evidence of a pleurisy that never presented distinct symptoms in life. Such pleurisy may be located in any part of the pleura but it is oftenest found at autopsy in the lung apices.

Etiology—It is generally agreed that acute fibrinous pleurisy is almost always a secondary inflammation. The primary inflammation may be in the lung, the diaphragm, the mediastinal or pericardial tissues, or even the abdominal organs. An infection in the upper dome of the liver frequently causes pleurisy. The most common primary infection that produces pleural inflammation is tuberculosis. Pneumococcal infections of the lungs also produce a secondary pleurisy. In most cases in which acute fibrinous pleurisy can safely be presumed to be primary without a precedent inflammation in some other part of the body, there is a history of trauma at the site.

Pathology—Any inflammation of a thoracic organ and particularly an inflammation of the lungs may produce a non-specific inflammatory reaction on adjacent pleural surfaces. This non-specific reaction is the exudation of a serum in which an abundance of fibrin and cellular and lymph elements is characteristic. Such serum is quite viscous so that normal respiratory movements produce friction and irritation on contiguous surfaces. This actually is the condition of acute fibrinous pleurisy.

The response of the organism to pleural pain is an attempt to eliminate the rubbing of irritated surfaces by immobilization of the pleura. This relative immobilization is favorable to the development of the adhesions and symphysis that are so frequently the permanent residue of acute

When the primary infection is tuberculous, caseating nodules may develop on the pleural surface, producing a kind of dry pleurisy. Likewise malignancy may produce metastatic nodules.

Clinical Symptoms—When there are presenting symptoms, they usually begin with a sudden sharp pain in the chest and occasionally with a chill and a slight rise in temperature. Such a sudden sharp pain is postulated to indicate involvement of the parietal pleura, since experimental studies indicate that while a visceral pleura is sensitive to some degree, only the parietal pleura is capable of producing sharp sensation. Pain will continue to be felt in breathing or whenever pleural movement is made. When the pleurisy involves the lower anterolateral region of the thorax, the pain may

of pain

A condition requiring some special note is pleurisy located on the dia

ACUTE FIBRINOUS PLEURISY

phragm It is marked by much more severe pain than other types of pleurisy and because it may be referred to organs of the abdomen, such as the gall bladder or kidney it sometimes leads to radical treatment that is unnecessary. It will often also present symptoms of pain in the neck or shoulder which serves to distinguish it from infection of the abdominal organs.

Physical Signs—Even where limitation of movement of the affected side is not noticed in inspection it will usually be detected by palpation. Local pressure over the inflamed area will increase the sensation of pain. The percussion note is ordinarily slightly higher probably due to increased muscle tension in the area. The most significant physical sign is a faint scratching friction rub that may be heard or felt over the area of the pleurisy. It will be more intense on forced inspiration and tends to increase in volume as other symptoms subside.

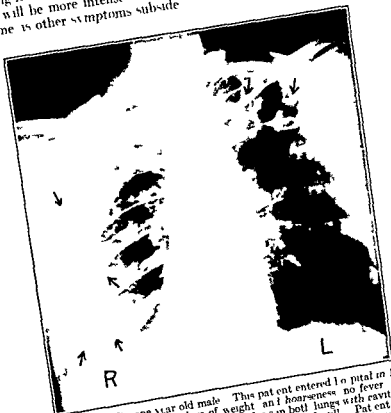


FIG 23 A F a fifty-one year old male. This patient entered hospital in 1934 with a history of cough expectoration loss of weight and hoarseness no fever Physical signs and roentgenogram showed definite tuberculous in both lungs with cavitation less cavitation of the left lung. Sputum was positive for acid fast bacilli. Patient was given pneumothorax treatment which was complicated by spontaneous pneumothorax followed by serofibrinous pleurisy. He later developed many lesions of the bones in both forearms and fingers and vertebra and in shoulder joints.

Roentgenogram (Bucky) 4-19-45. Right lung dense haze laterally extending from apex to diaphragm outlined by arrows. Discrete nodules throughout left lung cavity in upper lobe of left lung outlined by arrows. Suspicious cavity near larger cavity heart in midline.

Diagnosis: Bilateral pulmonary tuberculosis with left cavitation and fibrinous pleurisy.

Roentgen-ray Findings A roentgen ray examination will not give much information about a fibrinous pleurisy while it is still in the acute stage. If pneumothorax is used in the treatment of a primary condition such as tuberculosis a roentgenogram may show a slightly increased density of the pleura where it is inflamed.

After the acute pleurisy has subsided adhesions may be seen in the roentgenogram or indicated by abnormalities in the configuration of the lungs adhering to the pleura.

Where dry pleurisy is due to metastatic nodules these may sometimes be seen roentgenographically.

such direct vision will also reveal adhesions that were not indicated on the roentgenogram.

Differential Diagnosis—Since acute fibrinous pleurisy is in almost all cases a secondary condition the physician will be much less concerned with differentiating the pleurisy than with the identification of the primary condition. Adhesions may on a roentgenogram be momentarily confused for thin cystic walls but their characteristically straight lines should quickly indicate their nature.

Difficulty is most apt to be encountered in differentiating acute fibrinous pleurisy from metastatic malignancy. In malignancy the pain will usually

condition if it is not known.

BIBLIOGRAPHY

- BROWN S. Atypical forms of dry pleurisy. *radiolog. and clinical study* J. A. M. A. 102: 193, 1934.
CAPPS J. A. and COLEMAN G. H. An experimental and clinical study of pain in the

10. SEROFIBRINOUS PLEURISY

LIKE the dry form of fibrinous pleurisy serofibrinous pleurisy is rather benign and much of the time will clear up spontaneously it is more apt than the dry form however to develop into conditions requiring surgical intervention and thus its progress should be watched carefully It is not as frequently encountered as acute fibrinous pleurisy

Etiology—serofibrinous pleurisy seems to be in actuality an extension of the process of acute fibrinous pleurisy It is distinguished by the fact that the material exuded by the pleural surfaces in acute fibrinous pleurisy is exuded in sufficient quantity in serofibrinous pleurisy to form a free fluid As in the acute process it is almost always a secondary development it is most often secondary to tuberculosis It may however accompany almost any inflammatory condition of the lung or pleura, and may also accompany cysts and tumors It is occasionally due to an abscess of the liver to subphrenic abscess to subphrenic tumor or to a cancer of the kidney

Pathology Serofibrinous pleurisy begins like the acute type the pleural membranes lose their luster and are covered gradually with a deposit of fibrinous fluid The exudation continues until a free fluid straw-colored and usually clear has accumulated The amount may vary from a few cubic centimeters to several liters In a chronic case adhesions are likely to pocket such material and at times a cup-like or cascade formation of these pockets develops The primary abnormality may sometimes be resorption of blood and other substances being present in the fluid

In most cases the fluid is reabsorbed and the pleura regains its normal appearance In some patients it may be recurrent such cases however usually result in chronic pleurisy with fibrosis of the lung tissue developing from long compression Occasionally the serofibrinous pleurisy is a stage in the development of empyema seropurulent or purulent more often a chronic serofibrinous pleurisy will become infected and develop into an empyema

Symptoms Sometimes the onset of symptoms in serofibrinous pleurisy is abrupt beginning with a chill that is sometimes rather severe which is followed by fever and a sharp pain in the side The development may suggest lobar pneumonia

More often there is a history of slight cough lack of energy and loss of weight for several weeks These milder cases are characterized by moderate fever and pain The cough is usually dry Temperature continues to rise as long as the fluid accumulates but it is irregular Dyspnea may result from exertion if the effusion is massive As long as there is enough fluid present to lubricate the pleural surfaces breathing will not be painful

Physical Signs—The most important signs of fluid within the pleura are diminished movements of the thorax on the affected side with an increase in intercostal space flat sounds on percussion and diminished or absent breath sounds The absence of Litten's sign suggests the presence

of pleural fluid. If the effusion is basal Grocco's triangle of dullness will be elicited.

Tactile fremitus is usually diminished over a small effusion and absent over a large one. This is a more accurate method of determining the presence of fluid than percussion since by the latter method less than 400 cc is often undetectable.

In an unilateral effusion if the mediastinum is mobile the displacement of the heart toward the normal side takes place very early. The cardiac apex may be found as far over as the left axilla if the fluid is in the right cavity and to the right of the sternum if the fluid is on the left side.

If from 400 to 1000 cc of fluid is present percussion will elicit dullness. If the patient is sitting erect the fluid level can be mapped out as an

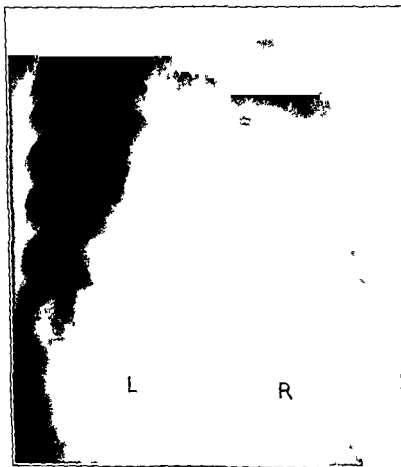
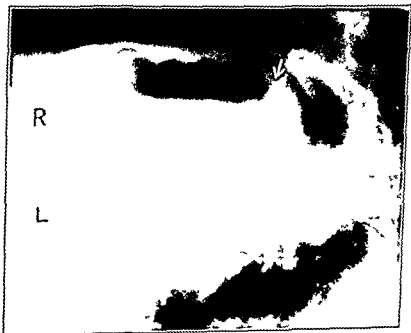


Fig. 24. Diaphragm in thorax. The roentgen ray film is in a lateral position in the right chest from the first rib and merging with the diaphragm of the liver. Diagnosis: Collapse of the lung. Serofibrinous pleurisy.



FIG. 2a — Same patient as shown in Figure 24. After a aspiration of fluid and replacement of air the size of the cavity is reduced. No the mediastinal densities are observed.
 Diagnosis: Carcinoma of lung. Serofibrinous pleurisy.



s shaped curve sometimes called Ellis curve. When enough fluid is present to cause solidification of the lungs due to compression bronchial breath and whisper sounds may be heard.

Roentgen ray Findings—From 300 to 400 cc. of fluid must be present for it to be visible in a roentgenogram taken in the normal anterior posterior position but much less can often be seen if a lateral roentgenogram is taken with the patient prone.

to the hilus of the lung

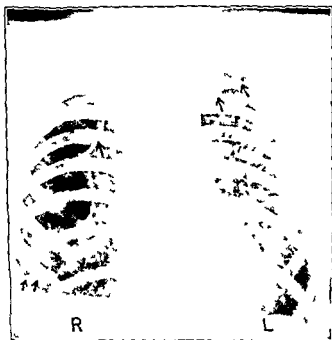


Fig. 10-11. Anterior-posterior and lateral views of chest with fluid in right pleural space. Left lung was normal.

Roentgen ray film 15-18. The right lung shows numerous thick lines suggesting cavities. They could be adhesions. A fluid level is noted in the lower right. Numerous small nodules throughout the right base are seen. In the left upper lobe a large cavity is seen. (Small arrows.)

Fig. 12. Bilateral tuberculous with adhesions, cavitations and fluid in pleural space.

Even a moderate amount of fluid will displace the mediastinum toward the normal side if it is not fixed by adhesions. Roentgen ray determination

of the extent of the fluid will be complicated if sufficient fluid is present to compress the lung since it is very difficult to differentiate between the shadows of the compressed lung the heart and the fluid unless iodized oil is injected into the tracheo bronchial tree. This latter procedure may occasionally be of considerable value in the diagnosis of pleural lesions.

When air and fluid are both present within the pleural cavity it will sometimes be of value to map the extent of the cavity and determine the extent of adhesions by changing the position of the patient before a fluoroscope and observing the shifting fluid levels. In the cascade type of serofibrinous pleurisy the different fluid levels become evident with changing of position.

Laboratory Findings—A pleural exudate will have a specific gravity of 1.018 or more, contain leukocytes and have a high albumen content. Other substances such as blood, chyle or cholesterol crystals may be indicated by the gross appearance of the fluid and suggest to the examiner the nature of the primary lesion.

Bacteriologic cultures should always be made of a serous effusion to determine if possible the nature of the primary inflammation. Sometimes a fungus infection will be indicated although the fungi themselves are seldom found in exudates in the pleura.

Where a large percentage of lymphocytes has been observed microscopically a check for tuberculosis should be made. The best test is guinea pig inoculation which will be more reliable if a large amount of fluid—500 to 1000 cc—is concentrated into a single dose. Several such inoculations should be made over quite a period of time since even in the presence of active tuberculosis the acid fast bacilli may be only irregularly present in the pleural fluid.

Particularly if the exudate has been bloody, malignancy should be considered. A Mandelbaum test as described in the first section of this book should be done. Again a single negative report is not decisive sometimes the fragments of tumor tissue and the isolated or bunched tumor cells do not appear in the microscopic examination of the fluid until the fourth or fifth aspiration.

Differential Diagnosis—The diagnostician has actually two problems in the presence of suspected serofibrinous pleurisy. The first problem is of course differentiation between the pleurisy and other conditions presenting similar appearances the second is the identification of the primary lesion.

The lesions most likely to be confused with serofibrinous pleurisy are the empyemas, hydrothorax or hemothorax, a thickened pleura, pneumonia, pericarditis with effusion, new growths, tuberculous empyema and occasionally subdiaphragmatic abnormalities.

Distinguishing a serofibrinous pleurisy from other pleural involvements is ordinarily done with certainty when the presence of a fluid is established by thoracentesis and a laboratory analysis has proven its serofibrinous character. However because the tapping of the chest wall is not without hazard the physician should establish his diagnosis on other bases if it is at all possible and confirm such findings with thoracentesis when it is indicated.

Empyema—An empyema will in most instances present a history of pneumonia or general sepsis that will indicate its nature. The clinical symptoms are roughly the same as those of serofibrinous pleurisy except that they are much more severe with higher temperature and greater loss of weight and strength. A secondary anemia develops rapidly, and a leukocytosis of 12 000 or more is present. Superficial veins are often dilated with an empyema.

As before remarked a serofibrinous effusion sometimes seems an inter-

the symptoms and their persistence will soon indicate the presence of a sepsis.

An effusion in a child less than five years old is almost invariably an empyema.

Hydrothorax and Hemothorax—Since hydrothorax is usually asymptomatic and since it is a secondary and latter development to some other major disturbance usually cardiac or renal disease, it will almost always be distinguishable on the basis of history alone. Further evidence of hydrothorax is found in the very slow accumulation of the fluid and its greater tendency to bilateral development. In most cases there will also be some edema of the extremities. Its nature as a transudate is easily determined by laboratory study.

Hemothorax results from damage to a vein or artery within the thorax. This damage is usually traumatic in origin and in such instances the hemothorax will offer no difficulty in diagnosis. Occasionally hemothorax is encountered in malignancy or advanced tuberculosis if the primary condition is not recognized which is unlikely a diagnostic puncture will be necessary to differentiate hemothorax from serofibrinous pleurisy or hydrothorax.

Pericarditis with Effusion—When there is a history of rheumatism associated with fluid in the left side of the chest, pericarditis with effusion is indicated. Further indications are cyanosis, urgent dyspnea, dilatation of the cervical veins and a paradoxical pulse. An electrocardiogram will show definite abnormalities.

The cardiac shadow on a roentgenogram will be oval or pear-shaped and may reach as high as the second rib. Percussion will elicit this distortion also.

When the patient is horizontal the area of sub-ternal dullness is less than when he is sitting upright. There will be dullness in the left infra-scapular region. Breathing will be bronchovesicular and tubular and a pericardial friction rub will be noticed when the fluid is minimal. The heart sounds will be faint. The cardiac impulse determined by auscultation since it will be too small to feel will be in its normal position which it would not be if the effusion were in the left pleura.

A diagnostic puncture into the pericardium will confirm the diagnosis. Replacement of some of the fluid with air will show a level in fluoroscopic studies and a roentgenogram in the various positions will outline the pericardium in its entirety.

Thickened Pleura—Occasionally roentgenographic studies may indicate fluid in the pleural cavity but no fluid will be obtained by diagnostic puncture. Iodized oil injected into the tracheo-bronchial tree, however, may indicate fibrosis of the pleura or the presence of a fibrin body in the pleura by revealing stenosis and resultant atelectasis in the suspect area.

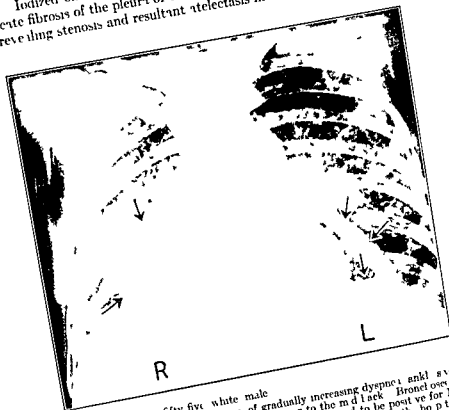


FIG. 28—J. M., age fifty-five, white male. Admitted May 12, 1945 with history of gradually increasing dyspnea, ankle swelling and episodic sharp anterior chest pains which were found to be positive for Mycobacterium tuberculosis when injected into a guinea pig. His course in the hospital was one of more or less constant dyspnea with a low grade fever. In August 1945 a pericardial friction rub was heard. Thereafter frequent pericardial aspirations were done.

Roentgenogram of the chest on December 7, 1945 showed fine granular densities distributed throughout both lung fields, some homogeneous density remaining over the lower part of the right lung field and a hydropneumopericardium with thickened parietal pericardium.

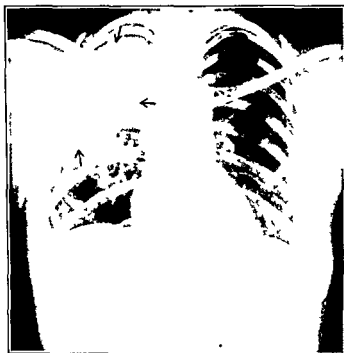
Autopsy on February 14, 1946 revealed pulmonary tuberculosis with lymphohematogenous dissemination, tuberculous pericarditis, tuberculous enteritis and perforation of the trachea and congestive heart failure.

Diagnosis: Hydropneumopericardium with thickened parietal pericardium, pulmonary tuberculosis.

Physical signs offer the best method of distinction. Percussion over the suspected area will elicit a note that while dull is not as dull as the sound over fluid. Breath sounds will be particularly faint with expiration which will be prolonged. Grocco's triangle will be absent and the heart will be in its normal position. (See Figure 23, page 62.)

Pneumonia —It is not infrequently difficult to differentiate serofibrinous effusion from an early pneumonia. Both are marked by some immobility during respiration and by thoracic pain. Pneumonia symptoms, however,

are overriding, a hard collection. The sputum, if present, and cultures of such sputum will usually provide conclusive evidence for diagnosis.



Difficulty is more likely to occur in the diagnosis of a fluid collection that arises during the course of pneumonia or at its subsidence. Since such fluid is most likely to be an empyema this differentiation will be discussed in that connection.

New Growths —Neither cysts nor neoplasms are often found in the pleura.

SEROFIBRINOUS PLEURISY

however both of them tend to be accompanied by serofibrinous pleurisy when they are found in that area.

The most common new growth that produces pleural effusions is lung malignancy with or without pleural involvement. Such malignancy will be suspected by the clinical picture it presents. Bloody sputum, stridor, dilatation of the pupils of the eye, dysphagia and distention of the veins of the neck all frequently accompany a malignancy of the lung.

Thoracentesis is necessary to the sure identification of a tumor or a cyst. The fluid aspirated will usually indicate a tumor by mulberry like masses of endothelial cells or microscopically by recognizable tumor cells. The effusion in connection with a malignancy will very often be bloody.

After thoracentesis the tumor unless it is a generalized metastasis or the cyst will usually be visible roentgenoscopically. Diagnostic pneumothorax is particularly valuable in distinguishing a cyst or tumor when it is accompanied by a pleural effusion. (See Figure 24, page 65)

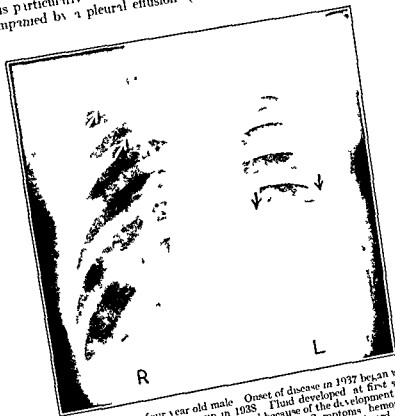


FIG 30 — J C a forty four year old male. Onset of disease in 1937 began with small hemoptysis. Left pneumothorax begun in 1938. Fluid developed at first serous and later frank pus. Pneumothorax was discontinued because of the development of a bronchial fistula. Sputum was positive for acid fast bacilli. Symptoms hemoptysis and weakness. Physical signs dullness over left chest, no breath sounds heard over right upper lobe. Breath sounds are bronchovesicular below clavicle they are cavernous. Roentgen ray film shows densities in right upper lobe with suggestion of cavity pneumothorax and fluid level in left.

Diagnosis Tuberculous empyema

Tuberculous Empyema—Serofibrinous pleurisy secondary to pulmonary tuberculosis is distinguished from tuberculous empyema by studies of the pleural fluid. The former is a non-specific reaction and hence acid-fast bacilli are usually undemonstrable or demonstrable only by culture or animal inoculation. Tuberculous empyema, on the other hand, will usually present grossly purulent fluid containing large numbers of leukocytes, especially lymphocytes, acid-fast bacilli are usually much easier to demonstrate. Mixed tuberculous empyema will show other bacteria in addition—pneumococci, streptococci, etc.

Subdiaphragmatic abnormalities—Occasionally a subphrenic abscess or tumor or an abscess of the liver must be excluded in a diagnosis of sero-fibrinous pleurisy. These conditions will be most often found on the right

found to be elevated by percussion or roentgen-ray study and dullness will be elicited over the lower right chest posteriorly. The heart will not be displaced. In an abscess of the liver a friction rub may be noted. (See Figure 28, page 70.)

BIBLIOGRAPHY

- BERLINER K. Hemorrhagic pleural effusions as an entity of 120 cases. *Ann Int Med* 14, 229th 1913
- BROCK R C and BLAIR I A. Importance of respiratory movements in formation and absorption of pleural fluids. *J Thoracic Surg* 1 150 1931
- GRAHAM F A. Influence of respiratory movements on formation of pleural exudates. *J A M A* 76 784 1921
- GRANT J A S. *Textbook of Surgery*. 1930
- GRANT J A S. *Surgical Diseases of the Chest*. 1930
- GRANT J A S. *Radiology* 45, 147 1945
- GRANT J A S. *with pleural effusions and ascites*
- GRANT J A S. *laboratory findings and clinical*
- GRANT J A S. *Am J*
- 1944
- 153 344
- SACRI, J. and RICHER, I. G. Mechanical pleural effusion, *Am J Roentgenol & Rad Therap* 24 225 1930
- SINGER J J. Diseases of the pleura. *Modern Medical Therapy in General Practice*. Ed. D. P. Barr, Baltimore. Maryland. Williams & Wilkins 1940 p 251
- SINGER, J J. Diseases of the pleura, *Text a Practice of Medicine*. Hagerstown. Maryland. W. F. Prior 1943 6 777
- THOMPSON H C. Pathogenesis of pleurisy with effusion. clinical epidemiological and follow up study of 190 cases. *Am Rev Tuberc* 54 311 1947
- FINNEY W S. and OLSEN A M. The significance of fluid in the pleural space. a study of 274 cases. *J Thoracic Surg* 14 218 1946

11. HYDROTHORAX

A discussion of hydrothorax is pertinent to a consideration of pleural pathology only because it presents many of the appearances of other abnormalities. It is a non-inflammatory secondary condition that seldom causes clinical symptoms and subsides when the primary disorder is rectified. In many cases treatment even simple aspiration is unnecessary.

Etiology—Hydrothorax results from edema of the pleura. The mechanism of an edema is not thoroughly understood. It is postulated that the edema fluid is exuded from endothelial cells either due to a pressure change in blood capillaries or to metabolic changes.

In any case this fluid since the pleural membrane has no penetration resisting envelope oozes slowly from the tissues of the pleura and accumulates. It is probable that edema affects the pleura to some extent in any of the dropsy producing conditions but the fluid is most apt to be present in noticeable quantities when the primary abnormality is a renal or a heart disease. A tumor which presses on the azygos vein and possibly thrombosis of that vein may also produce clinically important hydrothorax.

Meigs and Cass also point out the frequency with which hydrothorax and ascites in combination accompany an ovarian tumor. The etiology of this development is not at all clear.

Pathology—In hydrothorax the fluid is a transudate. It is yellow and clear and thin. The pleural surfaces will ordinarily remain quite normal in appearance occasionally when the hydrothorax is of long-standing they may be slightly cloudy and swollen.

Clinical Symptoms—In the great majority of patients hydrothorax will present no symptoms of itself. The possibility of symptoms exists only when the effusion is sufficiently massive to encroach upon the heart and lungs. In rare instances the resultant pressure symptoms may be quite severe even to complete atelectasis of the lung and concomitant cyanosis. Very often there is an associated edema of the lungs which gives rise to cough and sputum.

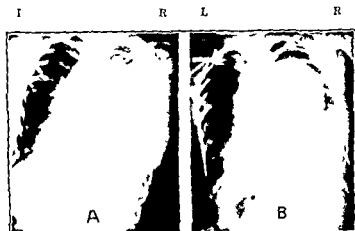
Physical Signs—The physical signs of hydrothorax are the same as those of serofibrinous pleurisy. The only difference to be noted is that hydrothorax is much more apt to be bilateral and that in almost all cases of such bilateral development the right side will contain much more fluid than the left.

Roentgen ray Findings—The only roentgenoscopic distinction between serofibrinous pleurisy and hydrothorax that might be noticed would be due to the lessened density of the transudate.

Laboratory Findings—The transudate of hydrothorax has a specific gravity of 1.015 or less. It is free from fibrin but may contain some red blood cells. Lymphocytes usually predominate early in the condition but they give way to polymorphonuclear leukocytes very soon. Albumin content is usually less than 1 per cent.

Differential Diagnosis—A hydrothorax is seldom difficult to differentiate from other conditions in the pleura. Only rarely as in the case of an ovarian

ian tumor or a constriction of the axillary vein, will it be a presenting symptom, normally the primary pathology will be noted before the hydrothorax and help identify it.

[illegible]

The presence of non-fluid lesions, such as fibrosis, cysts and tumors, will be diminished in the case of hydrothorax as they are for serofibrinous pleurisy. In most instances the transudate will be readily distinguished from the other fluids by its non-inflammatory character. A diagnostic thoracentesis whenever necessary will provide sure distinction.

BIBLIOGRAPHY

- Graham I. A., Singer J. J. and Ballou H. C.: Surgical Diseases of the Chest
J. Thor. & Gyner. 33: 243, 1937.
Nora E. D. and Davidson R. M.: Pleurisy with effusion associated with psoriasis re-
current cystadenoma (Meigs syndrome), Dis. of Chest 13: 423, 1947.
Packard J. S., and Gordon B.: Bilateral pleural collections of fluid, Am. J. Roent-
genol. 40: 859, 1938.

12. EMPYEMA (SUPPURATIVE PLEURISY)

when it occurs in children. Correct diagnosis is important since early treatment is necessary to prevent an acute empyema from becoming chronic or putrid.

Etiology Although empyema has been produced experimentally in the pleura by the use of irritants when it is found clinically it is bacteriological in origin. Occasionally it results from penetrating or crushing chest wounds but more often it is a sequel to infections of the lungs. Mediastinal or sub-diaphragmatic infection may also spread to the pleura. The most common micro organism in empyema is the pneumococcus, a particularly virulent form of the disease is occasioned by the streptococcus or the staphylococcus. Influenza bacilli especially the epidemic type may produce empyema. A rather lengthy list of micro organisms as a matter of fact have been established as occasionally producing this sepsis in the pleura. In many cases more than one organism will be present at the same time. The various fungi can also produce empyema.

Pathology—Empyema is characterized by the presence of pus in the pleural cavity. The amount is sometimes only a few cubic centimeters. On the other hand streptococcus hemolyticus in particular is apt to produce a purulent effusion that is massive but thin in consistency in the early stages.

The infection may develop in the pleural cavity by several routes. A pneumococcus infection which usually accompanies acute or subsiding lobar pneumonia can gain access to the pleural cavity through the finer alveoli. Streptococci which are usually the predominant organisms in lobular pneumonia or bronchopneumonia seem to enter the pleural space ordinarily through the interstitial lymphatics. Other micro-organisms may be blood borne or transmitted through the lymph channels. In the case of a chest wound of course the infection is direct and any infectious organism may gain entry by the rupture of an abscess of another organ into the pleural cavity.

In many cases the infection begins as a pyothorax with a thin greenish peptoid soup fluid that gradually becomes frank pus as the leukocyte content increases. However pneumococcal empyema the most common form of the disease. The latter form usually develops as a primary infection sometimes this infection may even appear coincidentally with the bronchopneumonia or the lobar pneumonia.

pyema however they may be very numerous not infrequently they will result in encapsulation of the fluid in either single or multiple pockets.

Spontaneous healing of an empyema rarely occurs and so almost inevitably a chronic form develops when an empyema is not recognized and properly treated. The improvement in treatment available in well-stuffed chest clinics in recent years has made chronic empyema a rather rare condition.

Clinical Symptoms—The clinical symptoms of empyema are usually overshadowed by the symptoms of the primary condition. Sometimes the development of a localized pain will indicate empyema but it is not to be depended on to announce the disease. Clinically the empyema should be considered as a possibility whenever fever and sepsis signs are present that are not readily attributable to some other condition or whenever such fever and sepsis seem excessive for the other condition.

The pneumococcus variety usually is ushered in as a sudden rise in temperature a few days to two weeks after the fever incident to pneumonia has subsided. In the past such a temperature rise has been attributed to unresolved pneumonia it is now generally recognized that such fever is almost always due to empyema.

Chronic empyema may be so far as sepsis signs are concerned almost symptomless. However there is generally evidence of long standing circulatory disturbance such as dyspnea edema or clubbing of the extremities. A chronic empyema often results in an empyema necessitatus which will be visible on the chest wall or it will rupture into the tracheo-bronchial tree and purulent material will be found in the sputum.

Any empyema if massive may produce dyspnea and pressure symptoms.

Physical Signs—The physical signs of empyema are essentially the same as those for scrofulous pleurisy which have been previously described. The helpfulness of physical signs to the diagnosis of empyema is minimized however by the fact that clinically important empyema may have so little exudate as to be undemonstrable by physical signs. The tendency to encapsulation further complicates the picture of empyema a small encapsulated empyema may give no physical evidence of itself at all.

Chronic empyema almost invariably results in an enormously thickened pleura that completely obliterates the signs of fluid when percussed.

Roentgen ray Findings—In the matter of roentgen ray studies empyema again is very similar to scrofulous pleurisy and again the limitations

so forth apply
only the same
quantities to

cast a shadow though it may be very difficult to identify the shadow as fluid roentgenoscopically. Particular difficulty is apt to be encountered with the rare condition of interlobar empyema which roentgenoscopically very closely simulates malignancy.

Chronic empyema is difficult to diagnose roentgenoscopically because of the characteristic fibrosis of the pleura. However when the empyema has been recognized the

Laboratory Diagnosis—A microscopic study of the purulent fluid is of course the decisive factor. Aspiration of a suspected empyema needs to be done carefully because bacteria in the needle track may spread the infection to the subcutaneous tissue and the structure of the chest wall, producing sinuses. The identification of the various micro organisms has become so routine as to require no discussion.

In acute empyema the changes in the blood will be typical of those accompanying any infectious process such as an increased leukocyte count and an increased sedimentation rate and the degree of change will be a measure of the seriousness of the empyema. In chronic empyema polymorphonuclear leukocytosis is almost always present, and a secondary anemia is also found in most instances.

Differential Diagnosis—The variety of roentgenologic appearances that empyemas may assume makes them easily confused with many other pathologic conditions. The most important ones are serofibrinous pleuritis, tuberculosis, and all of these conditions.

The best indication of an empyema other than the laboratory findings is a history of pneumonia since the percentage of empyemas that result from one or

1

physical signs or roentgenographic study suggest an empyema but such development is possible to pleurisy. Empyema is more often bilateral. Clinical symptoms may be quite similar. (See Figure 24, page 60.)

Hydrothorax—Hydrothorax is usually asymptomatic or at least it will not present the symptoms of sepsis that an empyema almost invariably does and it usually presents a history of renal or cardiac disease. Its fluid is always readily identifiable by the usual methods. (See Figure 31, page 75.)

Abscess of the Lung—In clinical symptoms abscess of the lung is very much like empyema but differentiation is usually possible on the basis of the patient's history. Abscess almost always is a sequela either to an operation, especially on the pharynx, or to the aspiration of a foreign body. Only rarely does it follow bronchopneumonia in which case the abscesses are usually quite small and multiple.

In size abscess of the lung seldom approaches an empyema although otherwise physical signs and roentgenoscopic appearances may be very much like. A large abscess located on the periphery of the lung may be

fluid may be helpful—the usual organism in an abscess is a coccus but it is not decisive, since both conditions may be produced by the same organisms.

The fact that an empyema may rupture into a lung or bronchus and that an abscess may likewise rupture into the pleural cavity and form an em-

pyemia complicates the diagnostic problem. Where the normal diagnostic procedures leave the physician in doubt, he is justified in following an aspiration and a diagnostic pneumothorax with thoracoscopy.

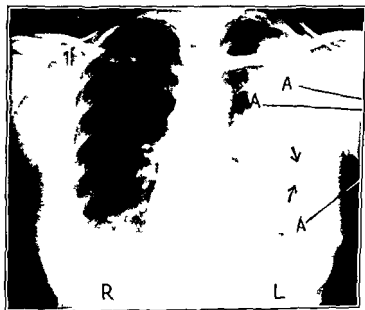
With an encysted interlobar empyema, distinction may not be made until an exploratory operation has been performed. (See Figure 105, page 208.)

Pneumonia — Any of the various pneumonias may sometimes resemble empyema in roentgenoscopic appearances. Lobar pneumonia is particularly apt to. Pneumonia is almost always indicated, however, by its physical signs of consolidation — bronchial breathing and increased whisper sounds over a large area of chest wall. Pneumonia also presents rather characteristic symptoms; the expectoration of rusty sputum is especially



There may often be difficulty in detecting the development of empyema as a secondary condition to pneumonia. It should be again emphasized that a sudden rise in fever following the abatement of lobar pneumonia usually indicates the development of an empyema.

Tuberculosis—Tuberculosis of the lungs will seldom be confused with empyema. The characteristic irregular densities in the upper lung with or without cavitation that are seen in a roentgenogram of tuberculosis are totally unlike the appearances of empyema. There is however an acute type of tuberculous pneumonia which gives an unilateral density that is much like that of a fluid, so the possibility of tuberculosis must be considered in a differential diagnosis of empyema. A diagnostic puncture into the pleural cavity will establish the presence or absence of empyema and sputum studies will usually demonstrate acid fast bacilli in tuberculosis.



of cases

with the treatment of

Tuberculous Empyema—A tuberculous empyema if it is not secondarily complicated by purulent micro-organisms may be practically asymptomatic on the other hand when it has become purulent it is usually more

violent symptomatically than the non tuberculous empyema. Differentiation can usually be made by the patient's history, otherwise the only basis of distinction is laboratory studies of the pleural fluid. The acid fast bacilli in an empyema are ordinarily only detectable by guinea pig inoculation (See Figure 30, page 72.)

New Growths — New growths will ordinarily be distinguished from empyemas as they are from serofibrinous pleurisy. The only special condition that needs mention is the interlobar empyema. In many instances only an exploratory operation will permit exact diagnosis. In fact it has not been uncommon for an operation for suspected carcinoma in this area to reveal that an empyema exists.



BIBLIOGRAPHY

- BRADY, F. D. Spontaneous empyema in children, *Am J Surg* 44, 122, 1933
- CANNON, H. T. and H. W. ...
- C
- L
- C
- C
- GRON, J. D. Chronic pleural empyema (its surgical treatment), *Dis of Chest* 12, 26, 1946
- HEAD, J. R. Treatment of empyema complicating artificial pneumothorax, *Illinois M J* 71, 493, 1937
- H
- H
- K
- O
- 1675, 1936
- peptidase activity of polymorphonuclear leucocytes, monocytes and epitheloid cells of pleural inflammatory exudates, *J Biol Chem* 125, 248, 1938
- WILLIAMS, M. H. Postpneumonic empyema in infants and children, treatment by open drainage, 39 cases, one death, *J Thoracic Surg*, 12, 160, 1912

13. PUTRID EMPYEMA

This empyema
at any age

occur

Etiology *anaerobes* particularly *streptococcus anaerobes*. Anaerobic cocci, staphylococci and fusiform bacilli are very common. Various aerobic bacteria are also present in some cases.

Putrid empyema is always a secondary development, usually to lung abscess. Oral infection seems also to play a prominent rôle in the etiology of the disease.

Pathology—Putrid empyema is usually an extension of a primary gangrenous condition of the lungs. The most common source of this infection is the rupture of a lung abscess into the pleural cavity; the bacteria may also gain access to the pleural cavity through a bronchopleural fistula in connection with long-standing bronchiectasis. Rarely putrid empyema occurs as a further complication to non putrid empyema that has been allowed to become chronic.

In any case putrid empyema is extremely virulent and leads to the death of the patient very rapidly unless positive surgical measures are taken.

Clinical Symptoms The onset of symptoms is usually quite sudden. They are in general those of pneumonia. Cough is more severe than the cough of a non putrid empyema, and chest pain is more severe and more generalized. If there is a bronchopleural fistula there may be foul sputum expectorated.

Physical Signs The physical signs are those of an acute non putrid empyema.

Roentgen ray Findings There is no way to distinguish putrid empyema and acute empyema roentgenologically. (See Fig. 30, page 72.)

Laboratory Tests Thoracentesis is the only way to definitely identify putrid empyema. The examiner will immediately recognize the condition by the foulness of the fluid, although laboratory smears may confirm the diagnosis and identify the particular anaerobe or anaerobes. Because infection of the needle track so often leads to general sepsis, it is important that the physician be in a position to perform an operation within twenty-four hours after the thoracentesis when putrid empyema is suspected.

Differential Diagnosis Putrid empyema will be differentiated from other pathological conditions just as the acute form is, when empyema is established; thoracentesis will differentiate between the putrid and non putrid. For differentiation from other conditions refer to illustrations and legends under *Empyema*.

BIBLIOGRAPHY

- BISGARD J D Skeletal deformities in children resulting from empyema and methods of prevention J Thoracic Surg 6 609 1937
- BLOXSON A P Empyema method of treatment with continuous irrigation and drain
- 1946
- HEAD J R Treatment of empyema complicating artificial pneumothorax Illinois M J 71 493 1937
- HEUER G J Acute empyema Pennsylvania M J 37 370 1931
- HOCHBERG L A Study of 300 cases of acute empyema thoracis 132 streptococci and 168 pneumococci J Thoracic Surg 10 354 1941
- KAUTZ F G and PINNER M Periapical empyema report of three cases with necropsy findings Am J Roentgenol & Rad Therap 37 446 1937
- OVERHOLT R H and BETTS R H Interlobar empyema S Clin North America 16 1675 1936
- RIGLER L G Roentgen study of mode of development of encapsulated interlobar effusions J Thoracic Surg 5 295 1936
- SCHWARTZ A O and MERRILL D M Chronic interlobar empyema J Thoracic

13. PUTRID EMPYEMA

PUTRID empyema deserves special mention not so much because of its frequency as because of its high mortality. Strieder and Lynch estimate that only between 5 and 10 per cent of all empyemas are putrid; the same authors, however, found in their series of 90 cases a mortality of 43 per cent. This empyema is primarily a disease of the elderly, although it may occur at any age. A heavy percentage of its victims are males.

Etiology—The characteristic micro-organisms of putrid empyemas are anaerobes, particularly streptococcus anaerobes. Anaerobic cocci, staphylococci and fusiform bacilli are very common. Various aerobic bacteria are also present in some cases.

Putrid empyema is always a secondary development, usually to lung abscess. Oral infection seems also to play a prominent role in the etiology of the disease.

Pathology—Putrid empyema is usually an extension of a primary gangrenous condition of the lungs. The most common source of this infection is the rupture of a lung abscess into the pleural cavity; the bacteria may also gain access to the pleural cavity through a bronchopleural fistula in connection with long-standing bronchiectasis. Rarely, putrid empyema occurs as a further complication to non-putrid empyema that has been allowed to become chronic.

In any case, putrid empyema is extremely virulent and leads to the death of the patient very rapidly unless positive surgical measures are taken.

Clinical Symptoms—The onset of symptoms is usually quite sudden. They are in general those of pneumonia. Cough is more severe than the cough of a non-putrid empyema, and chest pain is more severe and more generalized. If there is a bronchopleural fistula there may be foul sputum expectorated.

Physical Signs—The physical signs are those of an acute non-putrid empyema.

Roentgen ray Findings—There is no way to distinguish putrid empyema and acute empyema roentgenologically. (See Fig. 30, page 72.)

Laboratory Tests—Thoracentesis is the only way to definitely identify putrid empyema. The examiner will immediately recognize the condition by the foulness of the fluid, although laboratory smears may confirm the diagnosis and identify the particular anaerobe or anaerobes. Because infection of the needle track so often leads to general sepsis, it is important that the physician be in a position to perform an operation within twenty-four hours after the thoracentesis when putrid empyema is suspected.

Differential Diagnosis—Putrid empyema will be differentiated from other pathological conditions just as the acute form is, when empyema is established; thoracentesis will differentiate between the putrid and non-putrid. For differentiation from other conditions refer to illustrations and legends under *Empyema*.

BIBLIOGRAPHY

- BISGARD J D Skeletal deformities in children resulting from empyema and methods of prevention J Thoracic Surg 6 609 1937
- BLOVSOM A P Empyema method of treatment with continuous irrigation and drainage J Thoracic Surg 6 698 1937
- BURRELL L S T Causes and treatment of pleural effusion and empyema Brit J Tuberc 27 122 1933
- CABITT H J and HARRIS A J The treatment of empyema by drainage of the chest Cole D I and Dolley J L J Thoracic Surg 11 169 1941
- COLE D I and DOLLEY J L The treatment of empyema by drainage of the chest J Thoracic Surg 11 169 1941
- DOLLEY J L The treatment of empyema by drainage of the chest J Thoracic Surg 11 169 1941
- GAY F L The treatment of empyema by drainage of the chest J Thoracic Surg 11 169 1941
- HEAD J R Treatment of empyema complicating artificial pneumothorax Illinois M J 71 493 1937
- HEUER G J Acute empyema Pennsylvania M J 37 370 1934
- HOCHBERG L A Study of 300 cases of acute empyema thoracis 132 streptococci and 168 pneumococci J Thoracic Surg 10 354 1941
- KAUTZ F G and PINNER M Pericapsular empyema report of three cases with necropsy findings Am J Roentgenol & Rad Therap 37 446 1937
- OVERHOLT R H and BETTS R H Interlobar empyema S Clin North America 16 1675 1936
- RIGLER L G Roentgen study of mode of development of encapsulated interlobar effusions J Thoracic Surg 5 295 1936
- SINGLETON A O and MOORE R M Chronic nontuberculous empyema J Thoracic Surg 11 169 1941
- WILLIAMS M H Postpneumonic empyema in infants and children treatment by open drainage 39 cases one death J Thoracic Surg 12 160 1942
- peptidase activity of polymorphonuclear leucocytes monocytes and epithelial cells of pleural inflammatory exudates J Biol Chem 125 248 1938

13. PUTRID EMPYEMA

This empyema is primarily a disease of the elderly although it may occur at any age. A heavy percentage of its victims are males.

Etiology—The characteristic micro organisms of putrid empyemas are anaerobes particularly streptococcus anaerobes. Anaerobic cocci staphylococci and fusiform bacilli are very common. Various aerobic bacteria are also present in some cases.

Putrid empyema is always a secondary development usually to lung abscess. Oral infection seems also to play a prominent role in the etiology of the disease.

Pathology Putrid empyema is usually an extension of a primary pyogenic condition of the lungs. The most common source of this infection is the rupture of a lung abscess into the pleural cavity the bacteria may also gain access to the pleural cavity through a bronchopleural fistula in connection with long-standing bronchiectasis. Rarely putrid empyema occurs as a further complication to non putrid empyema that has been allowed to become chronic.

of

They are in general those of pneumonia. Cough is more severe than the cough of a non putrid empyema and chest pain is more severe and more generalized. If there is a bronchopleural fistula there may be foul sputum expectorated.

Physical Signs—The physical signs are those of an acute non putrid empyema.

Roentgen ray Findings—There is no way to distinguish putrid empyema and acute empyema roentgenologically. (See Fig 30 page 72.)

Laboratory Tests—Thoracentesis is the only way to definitely identify putrid empyema. The examiner will immediately recognize the condition by the foulness of the fluid although laboratory smears may confirm the diagnosis and identify the particular anaerobe or anaerobes. Because infection of the needle track so often leads to general sepsis it is important that the physician be in a position to perform an operation within twenty-four hours after the thoracentesis when putrid empyema is suspected.

Differential Diagnosis—Putrid empyema will be differentiated from other pathological conditions just as the acute form is when empyema is established thoracentesis will differentiate between the putrid and non putrid. For differentiation from other conditions refer to illustrations and legends under *Empyema*.

BIBLIOGRAPHY

- GRAHAM E A SINGER J J and
 delphis Lea &
 RUBIN E H and I
 1947 p 550
 SINGER J J Disc
 land W F Prior 1940 5 377
 STRIEDER, J W and LYNCH J P Putrid empyema New England J Med 234 1
 1946

14. TUBERCULOUS EMPYEMA

TUBERCULOUS empyema may be acute or chronic and it may be com-

Etiology—An active tuberculous infection in some other part of the body is always a precedent condition in tuberculous empyema. Occasionally this tuberculous condition is in a vertebra and the mechanical factor in transmission is the rupture of a cold abscess. In an overwhelming majority of cases however the primary infection is tuberculosis of the lung and the mechanical factor is the softening of a caseous nodule on the surface of the lung or pleura.

In a few instances the disease starts as a simple serofibrinous pleurisy and the disintegration of the softened nodule can be presumed to be without mechanical cause. In perhaps a fourth of them statistics from different institutions vary somewhat—spontaneous pneumothorax accompanies the softening of the nodule. In the majority of cases reported the softening of the nodule has occurred during the use of pneumothorax, pneumolysis or thorocoplasty in the treatment of tuberculosis.

Pathology—In the early stages the fluid of tuberculous empyema is usually thin and per soup in character in the later stages it is more apt to present the appearance of true pus. Sometimes the fluid is bloody or a long retained tuberculous exudate may appear opalescent due to cholesterol crystals.

A mixed tulero le sem e na most often cam sea sea lt f n

The pleura in tuberculous empyema often becomes massively inflamed and unless treatment is undertaken within a short time of onset it may become greatly thickened and inelastic. In many cases the diaphragm will react similarly. The original reaction to tuberculous empyema is apt to be highly toxic but this reaction which resembles an allergy usually disappears gradually.

th

2

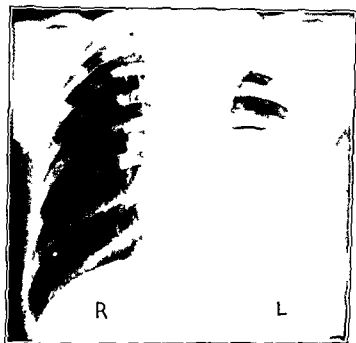
fir

$$p_0 \neq 0 \quad \text{if } n_0 \neq 1 \quad \text{and} \quad 1 \leq n_0$$

countered who is a symptomatic

Physical Signs In general the physical signs of tuberculous empyema are similar to those of any other fluid producing condition of the pleura

Roentgen ray Findings—The combination of fluid in the pleural cavity and tuberculous lesions in the upper lobes of the lung or collapse of the lung to some degree is rather good roentgenologic evidence of tuberculous empyema. It is not definitive evidence because tuberculosis of the lung may sometimes be accompanied by a simple serofibrinous pleurisy. The effusion in such pleurisy is usually small whereas a tuberculous empyema may be quite massive



Laboratory Tests By laboratory methods one can conclusively establish tuberculous empyema by the finding of leukocytosis in the pleural fluid in conjunction with acid fast bacilli. The detection of the bacilli in tuberculous empyema as in other pleural conditions is most certain with guinea pig inoculation and with cultures. The leukocyte count will not be as high ordinarily as in the non tuberculous empyemas. Lymphocytes will ordinarily be more numerous than polymorphonuclears.

Smears should also be made to see if streptococci, staphylococci or the other micro organisms of a non tuberculous empyema are present

TUBERCULOUS EMPYEMA

Differential Diagnosis—The differential procedure for tuberculous empyema is the same as for the other pleural fluid producing conditions. The various non fluid abnormalities are eliminated in the manner we have described in the differential section of the Serofibrinous Pleurisy chapter and tuberculous empyema is established by laboratory methods. The tuberculous nature of an empyema will usually be suggested by the patient's history of tuberculous infection but a simple empyema may coexist with tuberculosis. Final distinction is only possible by laboratory studies of pleural fluid. (See figures under Serofibrinous pleurisy and empyema.)

BIBLIOGRAPHY

- ALEXANDER J. The Surgery of Pulmonary Tuberculosis. Philadelphia: Lea & Febiger 1925.
- Charles C Thomas 1937.
- CORYLLOS P N. A new conception of tuberculous empyemata based on their pathologic physiology. J Thoracic Surg 7 48 1937.
- DONALDSON J H. Surgical Disorders of the Chest 2nd Ed. Philadelphia: Lea & Febiger 1947.
- FLEE F P and WYLIE R H. Empyema and unexpanded lung following artificial pneumothorax for tuberculosis. J Thoracic Surg 10 603 1941.
- GRAHAM F A, SINGER J J and BALLOU H C. Surgical Diseases of the Chest Philadelphia: Lea & Febiger 1935 p 163.
- ORNSTEIN G G and HERMAN M. Management of pure tuberculous and mixed infection empyemata. Dis of Chest 12 1 1946.
- SANGER P W. Decortication in acute empyema thoracis. Surg Gynec & Obst 82 71 1946.
- WOLFF W. Tuberculous empyema: report on 154 cases. J Thoracic Surg 7 420 1938.

15. SPONTANEOUS PNEUMOTHORAX

SPONTANEOUS pneumothorax is ordinarily classified as tuberculous or non tuberculous. The distinction is made on the basis of whether or not acid fast bacilli are in the pleura. A non tuberculous pneumothorax may occur during the treatment for tuberculosis through the rupture of adhe-

ture they are not consistent it is found more frequently in the larger chest clinics with extensive roentgen ray service than in general practice.

Tuberculous spontaneous pneumothorax may be associated with tuber-

ture may accompany any severe exertion but in most instances exertion does not seem to be a factor. Cases have been reported in medical literature in which spontaneous pneumothorax occurred due to hereditary weakness of the pleura but they are inconsiderable.

The rupture of an emphysematous bleb may also give rise to tuberculous spontaneous pneumothorax. More commonly the tuberculous variety of spontaneous pneumothorax is due to a caseous focus penetrating the pleura. The latter occurrence particularly may take place in a patient with tuberculosis that is not at all advanced. It occurs not uncommonly during treatment for tuberculosis and may complicate an artificial pneumothorax.

Pathology—The size of the tear in the lung varies widely of course with the pneumothorax produced so small as to be asymptomatic or so

non tuberculous kind such spontaneous closure occurs the air is absorbed and the chest cavity becomes normal. Frequently the resulting scar tissue will be weak however and there will be recurrent pneumothoraces and because blebs and bullae tend to be multiple pneumothorax may occur in another section of the pleura.

Where the tear is large spontaneous pneumothorax with partial collapse of the lung may persist until the tear is surgically closed. A complete collapse of the lung is uncommon. Occasionally a one-way valve will develop and a situation of tension pneumothorax will be created in which sufficient pressure will be gradually created to radically displace the thoracic organs.

The occurrence of fluid in the pleural cavity in conjunction with a spontaneous pneumothorax always suggests a tuberculous origin for the pneumothorax which can only be disproved by the most thorough laboratory examinations.

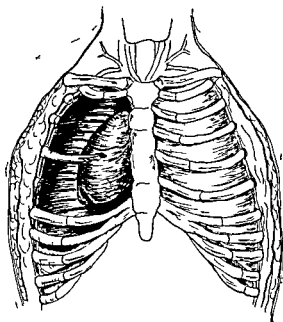
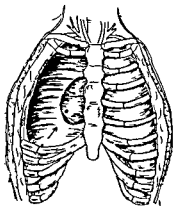
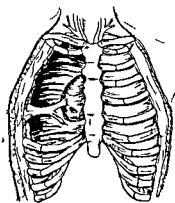


FIG 36 Diagrammatic drawing showing how a fractured rib can puncture the lung producing a traumatic spontaneous pneumothorax. Reprinted from *Texts & Lectures of Medicine* W. B. Saunders Company, Inc.



Clinical Symptoms —Symptoms will depend on the degree of lung collapse and the rate of the collapse. Spontaneous pneumothorax is rarely an asymptomatic condition discovered in routine examination. More often it manifests itself by gradually increasing symptoms of pain and heaviness in the chest. In the typical case the onset is sudden.

The chief symptom is pain, usually localized at the point of pleural tear although it may be referred to the shoulder or abdomen. As the air accumulates the patient will feel that his chest is being squeezed in a vise.



FIG. 35 — Spontaneous pneumothorax and numerous cysts of the right lung

When the attack is sudden the pain may resemble that of angina pectoris or coronary thrombosis and dyspnea may be extreme. In rare instances the pneumothorax has been so massive as to kill the patient by compression of the mediastinal vessel.

Shock is oftentimes a serious consideration when the onset of spontaneous pneumothorax symptoms is sudden. Pallor, sweats and unconsciousness are not uncommon.

Physical Signs —The physical signs vary with the amount of air in the chest cavity and also with the amount of time which has elapsed since the

pneumothorax occurred. The heart and mediastinum will be displaced

sounds will be distant or absent over the air pocket. If fluid is present it can



a flattened or even concave diaphragm will usually be immediately notice-

be shifted toward the contralateral side. Lung compression and mediastinal displacement will of course be especially noticeable if the condition is a tension pneumothorax.



Fig
phragm
exposed

Diagnosis

Large tuberculous cavity in left lung

with Backy dia
right lung is over

show a
The
ossible

the differentiation of a tuberculous from a non tuberculous spontaneous pneumothorax

If fluid is present Kienbock's phenomenon in which the diaphragm on the affected side rises on inspiration and falls on expiration may be noted. By changing the position of the patient before the fluoroscopic screen changing fluid levels can be noted and the pneumothorax pocket thus outlined. Waves of fluid can be demonstrated by slapping the chest of the patient or will be noted when he coughs.

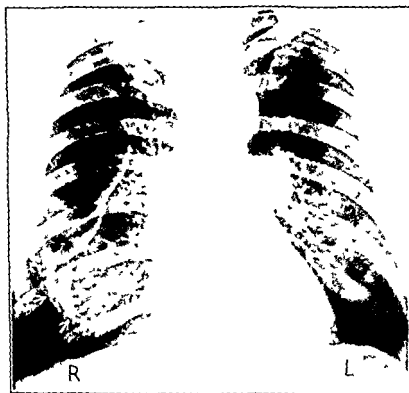


FIG. 41.—Acquired cysts of the right lung with emphysema of the entire left lung. There is a possibility that there is fluid in the interlobar space outlined by arrows. Arrows point to cyst walls.

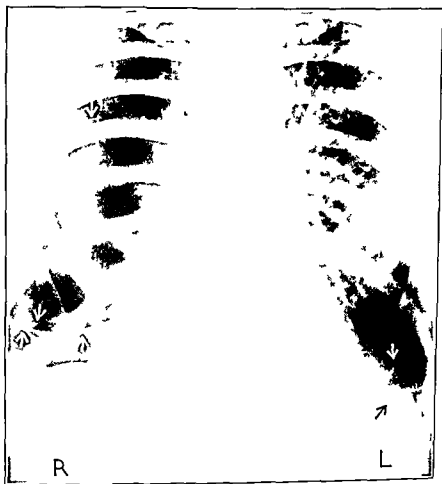
Diagnosis: Acquired cysts of the lung resembling post-traumatic pneumothorax.

Thoracentesis and Thoracoscopy Thoracentesis will be valuable in

a spontaneous pneumothorax. Where it is otherwise indicated however it is a useful means of locating the lesion and determining its extent and

oftentimes caseous nodules on the surface of the lung or pleura will indicate the tuberculous nature of the pneumothorax. Adhesions will also be visible.

Laboratory Tests In the presence of pneumothorax particularly if fluid is present extensive guinea pig inoculations of sputum concentrate and pleural fluid if any should be made for acid fast bacilli. Even where the adjacent lung appears completely free from tuberculous lesions roentgenoscopically these precautionary procedures should be followed.



Differential Diagnosis — In a differential diagnosis the physician should consider diaphragmatic hernia, cavitation of the lung, extensive emphysema of the lung, cysts and pneumoperitoneum.

Diaphragmatic Hernias — When such a hernia is suspected it can easily be verified by introducing barium into the stomach and then giving the

difficult to differentiate at times from a tuberculous spontaneous pneumothorax. Composition of the air in the pocket can be determined by diagnostic aspiration, if a larger percentage of CO_2 is present in the air pocket than is present in the atmosphere a tuberculous cavity is indicated. It is

R

L



FIG. 43 — Pneumoperitoneum with phrenic crush on the right side. Arrows point to the right diaphragm.

Diagnosis: Artificial pneumoperitoneum.

alternating positive and negative pressure shown with forced inspiration and expiration.

With a cavity, roentgenologic evidence of tuberculosis in other parts of the lung is almost invariable; such evidence may be present with tuberculous spontaneous pneumothorax but it is not always present. Therefore,

if such lesions are not seen the likelihood is that the air pocket is a pneumothorax (See Fig 40 page 92)

Emphysema of the Lung — An extensive emphysema of the lung may be mistaken for a spontaneous pneumothorax but when the patient is viewed at different projections before the fluoroscope the thin outline of the bullae can usually be seen. Also an emphysema will usually present a history of long standing dyspnea and the patient will usually have a somewhat barrel shaped chest (See Fig 42 page 94)

Cyst of the Lung — A cyst will likewise usually have characteristic markings when seen roentgenoscopically. Further roentgenologic evidence may be obtained by the introduction of iodized oil into the cyst cavity, the trabeculae common to cysts can easily be seen (See Fig 41 page 93)

Pneumoperitoneum — This condition by pushing the diaphragm high into the chest cavity may possibly confuse the examiner. Its existence will usually be suggested by a history of a ruptured stomach ulcer or duodenum or puncture wounds of the intestines. Roentgenographic examination in various projections will usually reveal the liver spleen or kidneys in the air pocket if the condition is a pneumoperitoneum. Artificial pneumoperitoneum is sometimes used in the treatment of tuberculosis of the lung (See Fig 43 page 95)

BIBLIOGRAPHY

- ABBOTT O A and RENAI LT DE OLIVEIRA H Spontaneous pneumothoraces occurring in patients undergoing peroral endoscopy J Thoracic Surg 14 453 1945
 BEATTY G A Benign spontaneous pneumothorax Dis of Chest 4 15 1938
 BLACKFORD S D Spontaneous pneumothorax in college students J A M A 113 737 1939
 ENGLEHARDT H T and DERBES V J Spontaneous pneumothorax and bronchial asthma Ann Int Med 21 711 1944
 GRAHAM F A SINGER J J and BALLON H C Surgical Dis of the Chest Philadelphia Lea & Febiger 1935 p 173
 HENNELL H and STEINBERG M F Tense pneumothorax treatment of chronic and recurrent forms by induction of chemical pleuritis Arch Int Med 63 648 1939
 LEACH J E Pneumothorax in young adult males descriptive statistics in 126 cases Arch Int Med 76 264 1945
 KJAERGAARD H Spontaneous pneumothorax in the apparently healthy Acta Med Scand nav Supplement 43 291 1941
 LORGE H J Etiology of idiopathic pneumothorax Am J M Sc 199 635 1940
 MOVITT F R SMITH J V and FLOESSER L Treatment of spontaneous pneumothorax Dis of Chest 13 221 1947
 O'BRIEN F J and BENSON C D Spontaneous pneumothorax complicating pulmonary tuberculosis—its incidence and mortality Tr Am Ther Soc 33, 131 1933
 LYON M D and CRANDALL W B Surgical treatment of recurrent idiopathic spontaneous pneumothorax J Thoracic Surg 10 566 1941

MEDIASTINUM

16. ANATOMY

THE mediastinum consists of an anterior and posterior compartment. The anterior mediastinum lies in front of the root of the lung, the posterior mediastinum behind the root of the lung.

In the mediastinum *supracardium*, the first layer under the sternum is loose fatty tissue which is in situ, for the most part, the remains of the thymus gland. The second layer is formed by the innominate veins and by the inferior thyroid veins and the internal mammary veins both of the latter emptying into the innominate veins. The next structure which is

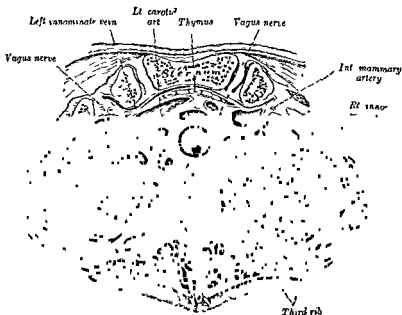
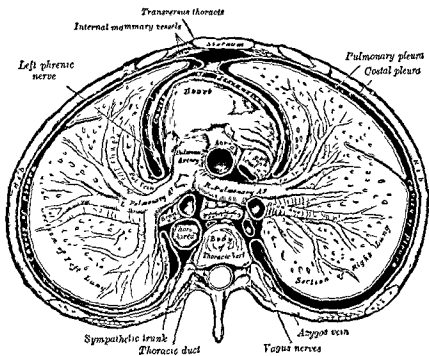


FIG. 41.—Transverse section through the upper margin of the second thoracic vertebra (Braune). The organs in posterior mediastinum are well outlined. (Gray's Anatomy.)

met and which lies for the most part in the midline, is the superior vena cava. The lower half of the superior vena cava is covered by pericardium and is therefore not visible. The great vessels, in reality, pierce the pericardium. The third layer is composed of the arch of the aorta and the branches which it gives off. The posterior portion and the fourth layer, composed of the trachea and the esophagus, form the anterior wall of the



posterior mediastinum Other structures to be considered in the posterior
the recurrent laryngeal nerves
pleura on either side of the
the nerves and medial to them



Fig. 46. M. scalenus medius from the right. 1. Esophagus and the esophageal

Julius Springer (Berlin)

BIBLIOGRAPHY

- CUNNINGHAM, D. J. Textbook of Anatomy, New York & London: Oxford University Press, 1943.
- BRUNS, H. Anatomie des Menschen, Berlin: Julius Springer, 1923.
- GRAY, H. Anatomy of the Human Body, 2nd Ed., Philadelphia: Lea & Febiger, 1918, p. 1127.

17. MEDIASTINITIS

BECAUSE of the intimate relationship of vital organs within the mediastinum inflammations in that area can be much more serious than the extent and severity of the inflammation would indicate. At the same time diagnosis of such inflammation is often very difficult.

For this discussion mediastinitis may be divided into phlegmons and abscesses. A non suppurative mediastinitis is also found rather frequently and can produce moderately severe symptoms of chills, fever, dyspnea and dysphagia, but since treatment is symptomatic and the condition usually subsides in a short time it will not be discussed.

Etiology The route of infection in mediastinitis has been the subject of much study in recent years. Pearse in a study of 110 cases found almost 60 per cent of them caused by trauma to the esophagus. This percentage is perhaps high, but there is little doubt that the major factor in mediastinitis is injury which permits the esophageal contents to leak into the mediastinum. Such injury is commonly due to the swallowing of sharp foreign bodies, such as bits of bone or glass. It may also occur during surgical instrumentation of the esophagus, particularly when there is a stricture of that organ. Carcinoma may perforate the esophagus when esophagoscopy is done in carcinomatous areas. Accidental tears are common.

Infection of the mediastinum is also rather frequently the result of the extension of cervical or upper respiratory infection. The route of such infection has been much debated; the various fasciae connecting the areas seem the most likely paths. The retro cervical space has also received attention in this connection.

node ulceration can represent an extension of the infection of a wide variety of organs.

Where mediastinitis is the result of esophageal tear, it is almost always

tinal collar abscesses.

A mediastinal abscess may undergo some fibrosis and become chronic, but the phlegmonous form of the acute disease is almost invariably too rapidly fatal to become chronic. Most instances of chronic mediastinitis

A chronic form of tuberculosis of the

Syphilis may progress through

on a widespread and mass the function of

mediastinal organs by mere mass. Phlegmonous mediastinitis has in the past run a usually very rapid and almost invariably fatal course since it was regarded as inoperable. Death sometimes followed within two days of the onset of symptoms. It is hoped, however, that the success that has attended the treatment of similar conditions with penicillin and streptomycin will make the prognosis of phlegmonous mediastinitis somewhat less grave in the future.

Although a mediastinal abscess lacks the limiting envelope that often encloses abscesses in other parts of the body, it is usually rather conservatively limited by inflammatory fibrosis. The character of fascial tissue also limits the infection. There is usually very little inflammation in surrounding tissues. In shape this abscess is usually a rounded cone and it frequently burrows in the direction that the tip points. It may rupture into bronchus, into the pleural cavity, or occasionally through the skin. Where drainage is established a spontaneous cure may result.

The most common site for both types of mediastinitis is the anterior mediastinum. Bilateral pleurisy is a not uncommon complication of mediastinitis, and either type may ulcerate through surrounding tissue.

Clinical Symptoms—Mediastinitis may manifest itself clinically in a great variety of ways. Dysphagia, fever and dyspnea are most commonly encountered. Edema and swelling of the face and neck may be noted, with cyanosis less likely. A sudden expectoration of a large amount of foul pus coincident with the rupture of an abscess into the tracheo-bronchial tree may herald spontaneous subsidence. More often, if there is an esophageal tear, small amounts of purulent, foul smelling pus will be expectorated at intervals.

Neuhof points out the sudden and deceptive remission of symptoms

when ulceration has occurred. Where ulceration has occurred, of course, the secondary site may be located by physical signs.

Roentgen ray Findings—The best indication of phlegmonous mediastinitis is an irregularly thickened central shadow in the chest. In many instances such an irregularity is difficult to detect. A film taken tangentially will usually indicate the condition most clearly. In some cases the central shadow will be seen to enlarge in successive roentgenograms within a very short space of time. A very strong sign of mediastinitis is a fluid level extending across the midline of the chest.

A mediastinal abscess may likewise be seen as an enlargement of the

the patient through many different angles before the fluoroscope before the shadow is made visible.

When mediastinitis is due to a tear of the esophagus, the lesion can be demonstrated and located by filling the esophagus with barium. A roentgenogram will then show the spill into the mediastinal area.

Laboratory Tests — Identification of the specific micro-organism may be made with sputum studies where there is a connection between the esophagus or bronchus and mediastinum. Such identification is important to treatment.

Differential Diagnosis — The most important distinction that needs to be made with regard to mediastinitis is whether it is phlegmonous or an abscess, since only the latter is subject to surgery. The author has tried to indicate the basis of this distinction which must be primarily based on



FIG. 47 — P. W., age forty-five, white male, ex. Admitted to the hospital on 4-19-46 with a six month history of productive cough and

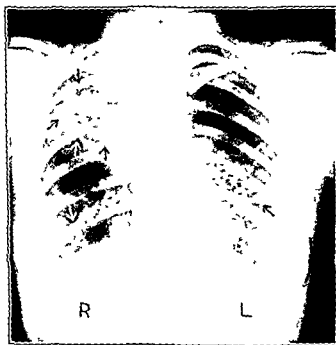
tinal emphysema.

Diagnosis — Mediastinitis with mediastinal and subcutaneous emphysema. Pulmonary tuberculosis.

Röntgen ray studies — In addition, the examiner may need to differentiate between mediastinitis and pneumonia, empyema, mediastinal tumors, and paraspinal abscess. Rarely, an aortic aneurysm may be mistaken for mediastinitis.



Fig. 48 — Acute clinical history suggested a diagnosis of atypical pneumonia and the serial roentgen ray films confirmed this diagnosis. Arrows outline pneumonic process.
 Diagnosis — Atypical pneumonia



Pneumonia — *Pneumonia* is usually identifiable by the finding of pneumococci in blood or sputum cultures. Where such findings are not made its characteristic temperature curve, the definite prodromal symptoms, the rusty sputum, the physical signs of consolidation, all serve to identify pneumonias with some certainty.

In roentgen ray studies a massive lung shadow, restricted to one lobe, identifies the lobar variety; bronchopneumonia will show irregular densities scattered through both lungs. Rarely is the central shadow enlarged as described for mediastinitis. (See Figure 48, page 103.)

R

L

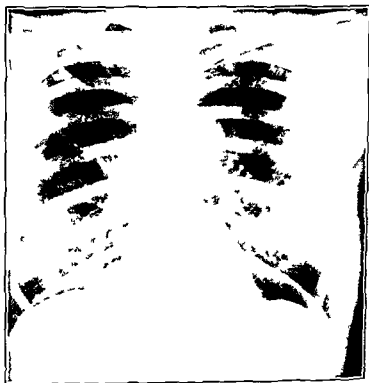


FIG. 50. Tumor of the mediastinum resembling abscess. Note shadow to left of spine at level of 2nd and 3rd anterior ribs.
Diagnosis: Mediastinal tumor (malignant thymoma).

Emphysema — A recent history of pneumonia or tuberculosis usually in the upper lobes, and a recent mediastinal area it is very common. In roentgen ray films, the mediastinal origin of the shadow will be revealed in following roentgenograms. This method may also

With either condition laboratory tests should be undertaken to determine the specific micro-organism, treatment, usually surgical, will be the same for both empyema and mediastinal abscess

Tumors — Mediastinal tumors, particularly the rounded benign varieties

toms as an abscess, but there will rarely be evidence of fever and toxemia such as characterizes an abscess

When diagnostic puncture is feasible the presence or absence of fluid can be determined, if the shadow is a tumor, the needle will frequently pick up shreds of tissue that will identify it

Paraspinal Abscess — Paraspinal abscesses are almost always tuberculous in nature. In a posterior-anterior view the roentgenologic appearance of such an abscess is indistinguishable from the appearance of mediastinal abscess. In a lateral view, however, the paraspinal abscess will usually be seen as an elongated, elliptical shadow hugging the spinal column. The mediastinum will be seen to be clear. Further evidence of the nature of a paraspinal "cold" abscess will be the erosion of the bodies of the vertebrae (See Figure 136, page 275)

BIBLIOGRAPHY

1. NEUBOR, H. and JEMERIN, F. E. Acute Infections of the Mediastinum, Baltimore: Williams & Wilkins, 1943

18. TUMORS OF THE MEDIASTINUM

EXCEPT for the lymph tumors, which will be discussed elsewhere most tumors found in the mediastinum are benign. Nevertheless it is generally recognized that such benign tumors have the possibility of malignant degeneration and that even if they do not become malignant they may eventually cause the death of the patient by interference with vital functions.

No growth in the mediastinum is of course out of place in a book of this nature. Mediastinal tumors however have a few characteristics that should be mentioned. Teratomas and dermoid cysts

Pathologists suggest that neurogenic tumors have their origin in nerve cells that have migrated from the ganglionic crest in embryonic life.

Although fibromas, chondromas and myxomas have been reported in the mediastinum only the lipoma occurs with any frequency. The etiology

The thymus itself is the origin of an interesting group of tumors including the primary carcinoma found most often with any frequency in the mediastinum. Sarcoma is also a possible development in the thymus. Certain

Thyroid tumors of the breast or even lower abdomen. Any carcinoma in this area other than cancer of the thymus should be suspected of being metastatic.

Pathology — The dermoid cyst consists of epidermal cells surrounding sebaceous material. Teratomas are extremely varied in structure they may contain all forms of tissue arising from mesenchyma. Both growths may

cede it. It is generally thought that teratoma is potentially malignant but a true dermoid cyst is the only growth in the mediastinum that cannot become malignant.

Neurogenic tumors may be made up of any type of nerve cell. They arise in almost all cases in the posterior mediastinum and because that region has more free space than the anterior mediastinum neurogenic tumors are frequently quite large when they are discovered. They have a tendency to sarcomatous degeneration. Neurofibroma perhaps as common as all the rest put together usually occurs as a single encapsulated tumor of irregular outline but tending to be round or oval. Microscopically it con-

sists of small round and fusiform cells which lie in a delicate reticulum

cells almost invariably predominate to give the tumor the characteristic soft consistency. Lipomas do not ulcerate into thoracic organs but they may spread widely through the chest cavity between the organs and become very difficult to remove completely. Partial extirpation is dangerous because suppuration often follows such a procedure. Lipomas may degenerate into liposarcomas.

Thymic tumors are found in the anterior mediastinum usually in the neighborhood of the sternal notch. Unless the tumor grows very rapidly fibrosis takes place and so therefore thymic tumors are usually among the hardest tumors found in the mediastinum. They are usually lobulated in form. The most common types are the lymphosarcoma and thymoma but occasionally a thymocarcinoma is found—the only primary carcinoma among the mediastinal tumors. The cellular structure that may be found in thymic tumors is extremely varied. A rather common feature of malignant thymic tumors is their tendency to ensheath blood vessels in the vicinity.

Clinical Symptoms Clinical symptoms will of course depend on the location of the tumor. The most common symptoms of both benign and malignant tumors are as might be expected symptoms of pressure. Edema of the face and neck is very common; it occurs when the tumor compresses the superior vena cava. In a few instances extreme cyanosis has been encountered. Dyspnea is also common and may be severe when a tumor has blocked or pinched off a bronchus and atelectasis has developed in a lung. Dysphagia is not unusual. The clinical symptoms of a tumor are apt to be intermittent.

Pain and loss of weight. Aggravation is an important location is an important a dull ache at the site of referred to other parts of tenderness to the touch.

Pain may also accompany a benign tumor if it compresses mediastinal organs. A tumor in the anterior mediastinum is most apt to be painful. Benign growths in the posterior mediastinum may achieve large size and be asymptomatic. Lipomas because they are usually quite soft may be asymptomatic even when they are in close juxtaposition to mediastinal organs. Dermoid cysts are the only growth in which a patient may sometimes cough up hair.

Physical Signs—Significant physical signs are apt to be difficult to elicit in the case of tumors. Where atelectasis occurs due to pressure on bronchi it can be noted by percussion and changes in breath sounds. When pleural effusion complicates a tumor the usual signs of fluid will be noted.

Such signs are however indications of complicating factors that may be produced by a great number of abnormalities

Dilated superficial veins of the lower chest wall may sometimes be observed. These may develop as a result of obstruction of the venæ cavæ as a form of col-

a malignan-

sternum

mass. In

diagnosis of mediastinal tumor

Roentgen ray Findings—Roentgenographic study is the only method by which one can be reasonably positive in the diagnosis of a mediastinal

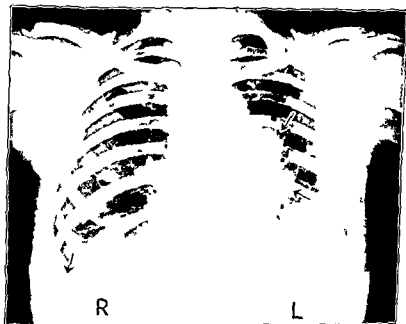


Fig. 1. Roentgen ray of the thorax. (From Dr.

1 op-

both

read

5 2

tumor. For almost every tumor in the area, however, a lateral view is a vital adjunct to the usual posterior-anterior view because of the interference produced by the cardiac shadow. Localization of a mediastinal tumor is most exact with a true lateral and a true posterior-anterior view. Where the tumor is large enough to encroach upon a lung, the injection of iodized oil into the tracheo-bronchial tree will at times outline the lungs in such a way as to make the mediastinal growth more prominent. It will also

indicate the degree of encroachment. When studying a tumor roentgenographically, it is to be remembered that a mediastinal tumor commonly grows to one side or the other so that it may appear in the film to be a lung tumor or a tumor of the chest wall and still have its origin in the mediastinum.

Lipomas may rarely be recognized roentgenoscopically by a peculiar halo-effect. Most benign tumors will suggest their nature by sharp outline. Teratomas will frequently contain identifiable bone or teeth that will be obvious on a roentgenogram especially when the Bucks diaphragm technic is used. Neurofibroma will be suggested by erosion of nearby bone.



FIG. 10. A. Case 1.

the area and respond most readily to such treatment. Sarcoma, the most common malignant growth in the mediastinum, is also radio sensitive.

Laboratory Tests—The exact nature of a tumor should be ascertained by laboratory methods wherever possible. Many mediastinal tumors, however, are so located that aspiration would involve lung puncture, which is dangerous. Where a tumor of the mediastinal lymph nodes is suspected, confirmation can sometimes be obtained by biopsy of the more accessible lymph nodes of the cervical region or other superficial lymph nodes.



FIG. 53. Lateral view of same patient as in Figure 52. The tumor shadow is outlined with arrows.

Diagnosis—Neurofibroma of the posterior mediastinum.

Too much reliance cannot be placed in a biopsy finding of benignity, since some tumors, particularly teratomas, may be completely benign in one section and malignant in another.

It is, of course, of course

is visible. Esophagoscopy will perform essentially the same service in connection with tumors impinging on the esophagus. There is some risk to the use of these methods because the bronchial or esophageal walls may be thinned by invasion by the tumor making perforation of the walls by the instrument likely. Thoracoscopy is frequently useful in diagnosis of tumors of the lower mediastinum. In many cases the direct view provided will indicate the

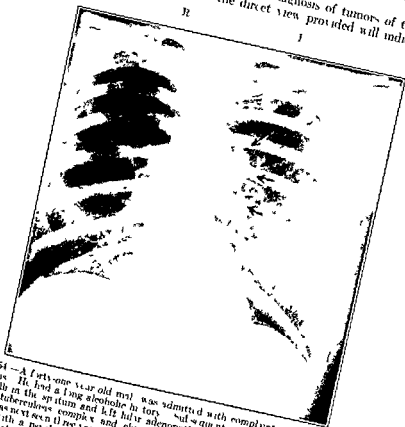


FIG. 54.—A forty-one year old male was admitted with complaints of weakness and flatulence. He had a long alcoholic history. Subsequent examination revealed no primary tuberculous complex and chronic alcoholism. Findings were interpreted as a patient was next seen three years later at which time he gave a history of dyspnea associated with a psychopathic personality. Died five days after hospital entry from cerebral metastases from the malignant thymoma. Roentgenograms 12-30-42 seen in the posterior anterior and left anterior oblique projections disclosed an oval uniformly dense mass with sharply defined margins lying in the anterior mediastinum to the left of the mid line at the level of the aortic arch—see arrows. Autopsy revealed a $17 \times 8 \times 7$ cm. tumor occupying the entire anterior mediastinum. Final diagnosis: malignant thymoma. (See Figure 59, page 110)

type of tumor and a biopsy specimen may be secured to confirm such indication. The degree to which the tumor adheres to surrounding organs an important consideration in surgery. It is sometimes seen. If the growth is malignant the pleura should be studied for metastatic nodules.

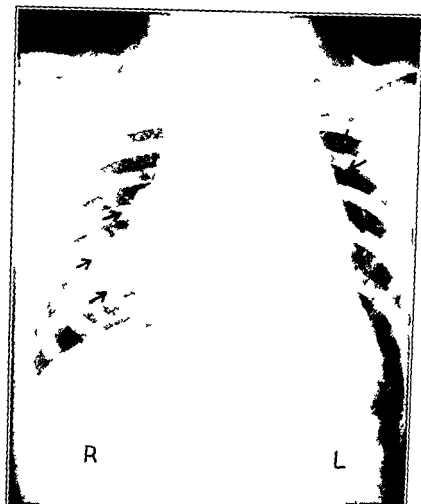
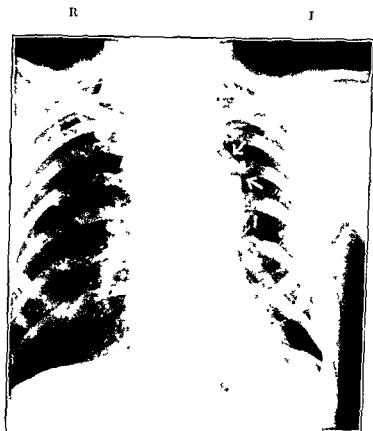




FIG. 50.—Lateral view of same patient as Figure 49 shows how the esophagus has been compressed by a tumor mass.
Diagnosis—Teratoma of the mediastinum.



¹ Autopsy diagnosis: Reticulum cell sarcoma of mediastinum with generalized metastases

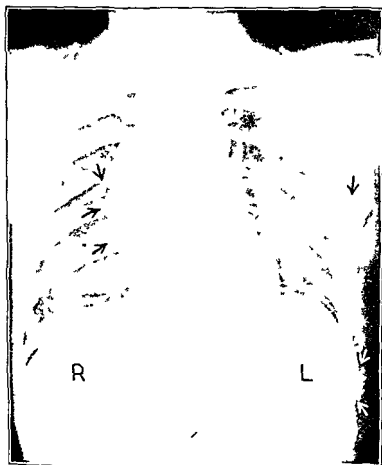


FIG. 58.—R I fifty five year old white male patient. Patient entered hospital with a history of pain in right leg for three years and recent and increasing

Differential Diagnosis—Diagnosis of an abnormality in the mediastinum will usually be difficult because of the involved nature of the area and the confusing roentgenographic appearances. A tumor of the mediastinum may be confused with a mediastinal abscess, dilatation of the esophagus, aneurysm of the aorta, aberrant thyroid, empyema or lymphoma.

Abscess—A mediastinal abscess will sometimes present a conical appearance on a roentgenogram but where it does not it may very much resemble



FIG. 59. Roentgenogram taken in tangential view of same patient as Figure 54 (Left anterior oblique roentgenogram).
Diagnosis—Malignant thymoma.

a tumor. Pressure symptoms may be the same for both conditions. An abscess, however, will usually present fever and toxemia, and the patient will also have a recent history of esophageal damage or infection in the cervical area. If diagnostic puncture is possible, differential diagnosis can be made with assurance.

Dilatation of the Esophagus—Cardiospasm may sometimes produce a dilatation of the esophagus that resembles a tumor. There may be mild pressure symptoms in conjunction with pronounced dysphagia and failure to retain food. In some cases loss of weight may be so striking as to suggest carcinoma. A diagnosis of dilatation can be made by a fluoroscopic examination following a barium meal.



Fig 60 - F I forty x year old mal ill ss leg n n 134 w tl ner a ng w ak
n uning lts cat no gl r f er A l g o w f t u b e l s w
k n P t t w a t l i h e d t l a r l u t e l
c t o r a t n s l g h t y t g d w t l l o o d o c h a l f
l h a l g n d n l e d n e n n a n d
l b e s L a t r l l b k f i l m l w t l d n t
S p c u f l u d l e c l n o t d a t b l k a r n w a

d n n l d t r n n tly n m d s e t u n a
(the al see) app n tly n m d s e t u n a
D agno s i n g a l c e s s



FIG. 61. C. I., male, age thirty. Three year history of difficulty in swallowing. The patient states that ingested substances seem to lodge at a point near the lower edge of the sternum. Liquids are obstructed to the same degree as solids. Roentgen ray examination of the

Diagnosis Cardio pasm

Aneurysm — An aneurysm of the aorta may be confused with tumor roentgenologically and symptomatically. However the aneurysm will usually present localized pain that is much more persistent than the pain of a tumor. It is more likely to extensively erode nearby bone. Pulsation the most characteristic feature of an aneurysm will be visible during the course of a roentgenologic examination particularly if a roentgenkymogram is taken. In some cases a lipoma may also transmit a pulse from nearby vessels but this weakened uncertain transmission will not be confused with an aneurysm. Plain films and kymograms will sometimes be helpful (See figure 61 page 121)

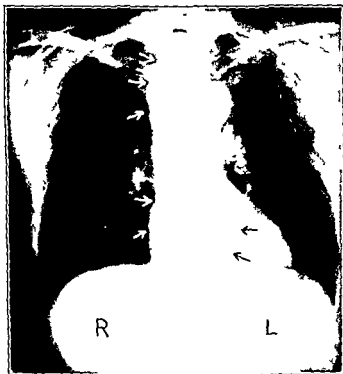


FIG. 62 — Aetiology of the esophagus. Posterior-anterior view. Arrows all to the wall of esophageal lumen.

Aberrant Thyroid — An enlarged thyroid gland may extend down into the subternal space where it may be mistaken for a tumor. If it reaches sufficient size it may even present the pressure symptoms of a tumor. In instances of a marked enlargement or cancer of the gland one may find Horner's syndrome caused by pressure on nerves. A metabolism test may suggest an aberrant thyroid but it is not by any means a sure basis for differentiation. However a roentgen ray examination and fluoroscope will usually indicate the nature of the abnormality by exactly localizing it.

In most cases palpable enlargement in the cervical area will provide further indication of thyroid enlargement. In rare cases an exploratory operation may be necessary to differentiate a tumor of the thyroid from other mediastinal masses. (See Figure 65 page 122.)

Empyema—Where a roentgenographic shadow indicates either a me-



FIG. 63.—Same patient in oblique position as in Figure 64. Note the distention of the esophagus by barium filling.
Diagnosis: Adenoma of the esophagus.

most instances. A conclusive diagnosis can be made if a diagnostic puncture is practicable. Instances have been reported in which positive diagnosis was only possible by an exploratory operation. (See Figure 30 page 72.)

Lymphoma—This condition will be suggested by the location of the suspicious growth in the area of the lymph nodes. There may be fever or constitutional symptoms of weight loss, weakness, etc. Biopsy of a cervical lymph node if available will make differentiation possible.



FIG. 64.—Compression of the esophagus by aortic aneurysm. I. I. male, age forty-five. This patient has a large aneurysm of the ascending portion and arch of the aorta. Roentgen ray examination after the ingestion of thick barium paste reveals compression of the lumen of the middle third of the esophagus by this aneurysm at A and arrow.

Diagnosis. Aneurysm compressing esophagus.



arch dilatation

Diagnosis Thyroid enlargement Substernal thyroid

BIBLIOGRAPHY

- ADAMS, W. F., and THORNTON, T. F., JR. Bronchogenic cysts of mediastinum with report of three cases. *J Thoracic Surg* **12**, 503, 1943.
- BELL, E. T. Tumors of the thymus in myasthenia gravis. *J Nerv & Ment Dis* **45**, 130, 1917.
- BELL, E. T. *Myasthenia Gravis*. New York: McGraw-Hill, 1924.
- BROWN, A. L. Complete occlusion of superior vena cava by primary carcinoma of the lung. *Arch Surg* **21**, 959, 1930.
- CARLSON, H. A. Congenital cysts of mediastinum, report of three cases including gastric cyst. *J Thoracic Surg* **12**, 376, 1943.
- CROSBY, I. *Case Report*. *J Med Case, J*
- CURTIS, G. T. *Case Report*. *Cancer* **16**, 461, 1932.
- FELDMAN, I. *Case Report*. *J* **29**, 549, 1930.
- FELDMAN, I. *Case Report*. *New York Path Soc* **23**, 47, 1923.
- FRANK, J. *Neoplastic Diseases*. Philadelphia: W. B. Saunders Co., 1928.
- MIXTER, C. G., and CLIFFORD, S. H. Congenital mediastinal cysts of gastrogenic and bronchogenic origin. *J Thoracic Surg* **13**, 161, 1944.
- ROBERT, N. L. Dermoid cysts and teratomata of mediastinum, review. *J Thoracic Surg* **13**, 161, 1944.
- SCHWARTZ, A. *Case Report*. *J* **43**, 115, 1944.
- SINGER, J. *Case Report*. *J* **313**, 1944.
- STOUT, A. P. Ganglioneuroma of the cervical and thoracic sympathetic ganglions. *J A M A* **82**, 1770, 1924.
- WATSON, W. L., and URRAN, J. A. Mediastinal lipoma: case report. *J Thoracic Surg* **13**, 16, 1944.

19.

Grossly it is seen

The diaphragm is a muscular partition separating the thoracic and abdominal cavities. It is covered by serous membranes on both sides, forming the pericardial and peritoneal cavities. The central tendon is a large, triangular, and extremely tough structure, supported by the pericardium above and the peritoneum below. It consists of three leaves: the center leaf, the right leaf, and the left leaf. The length of the fiber origin to this tendon is determined by the distance between the various muscle fibers of the diaphragm. The length of the fiber origin to this tendon is determined by the distance between the various muscle fibers of the diaphragm. The length of the fiber origin to this tendon is determined by the distance between the various muscle fibers of the diaphragm.

This text is directed to an understanding of diaphragm function with emphasis on the relationship of structure to physiological processes. The anatomical description has been drawn in part from various sections of Gray's *anatomy* as well as from work by Millar and other notable anatomists.

juncture This milking action is the primary aid to gravity in the swallowing mechanism and also is effective in preventing regurgitation

In a fluoroscopic examination of the thoracic cage the action of the dia

phragm and increase its excursion A quick and reasonably accurate check of diaphragmatic function is the observation of the progress of barium down the esophagus Any pronounced pathologic condition of the diaphragm will usually inhibit the normal milking action of the esophageal hiatus

There is a diaphragm that may exist without the right diaphragm is foremost shadow most prominent upon deep inspiration may be due to partial interference with nerve supply or a disease process of the lung causing unequal aeration Unless clinical symptoms or other evidence indicates diaphragmatic malfunction or inflammation it may be regarded as a purely physiologic anomaly

The costal components of the diaphragmatic attachment sometimes They expan

Not infrequently a tenting of the diaphragm is noticed upon deep inspiration which may resemble an adhesion This phenomenon is or finally found in the presence of a chronic lung inflammation and is thought to represent an inelastic action of the lung unable to follow the diaphragm upon inspiration and perhaps a physiologic weakness of the diaphragm at that point It can be distinguished from adhesions by a tendency to disappear or to be inconstant

Sometimes before the fluoroscope the right diaphragm will be seen to pulsate This pulsation is thought to be transmitted from the liver Pulsatory movements may also result from adhesions connecting the diaphragm and pericardium

The diaphragm is remarkably free from primary pathologic disturbances It may however be secondarily affected Impaired movement may result from adhesions or may accompany lung infections Any interference with the phrenic nerves of course surgical or pathologic may paralyze the diaphragm or may cause its paradoxical movement

Primary tumors and cysts of the diaphragm are uncommon sarcomata and endotheliomas have been reported but they are rare However meta

the chest cavity. Because of its rarity, its etiology has not been defined.



FIG. 67. B. M. male, ag. seventy. This patient gives a history of at least ten years

Neuromuscular degeneration because of neuritis or damage to the phrenic nerve has been suggested. But the fact that the condition is found without pronounced digestive or respiratory malfunction suggests that the condition is a congenital one to which the individual has been accustomed from birth. The left leaf is most often the deficient one. Symptoms are rare, and at all

20. DIAPHRAGMATIC HERNIA

BECAUSE the term diaphragmatic hernia implies merely the protrusion of an abdominal organ or part of an organ through the diaphragm the subject is in medical literature somewhat disorganized. The simplest division is into traumatic and non traumatic. Even this division however is somewhat arbitrary since congenital weakness may be a factor in a hernia directly caused by abdominal compression and since muscular strain or

hernia comparatively simple. The large number of congenital defects encountered in children usually in conjunction with other organic defects is a problem for the pediatrician and largely outside the scope of this work.

Etiology—It is now generally recognized that herniation not due to

lifting are accomplished by a further increase of the already positive intra-abdominal pressure. Hence any embryonic defect may at any time become

costal and crural fibers the diaphragmatic dome particularly the left and

through the hiatus.

Pathology—A weakness in the diaphragm once established the possibilities for displacement are extensive. At times practically the entire abdominal contents may be drawn up into the chest cavity with consequent radical displacement of the heart and mediastinum and complete collapse of one lung. At other times only a small portion of the abdominal contents is involved and the

the weakened structure is the esophageal hiatus the common finding is either the presence of the small end of the stomach in the thoracic cage with a shortened esophagus or the esophagus entering the abdominal cavity normally with a fold of the stomach thrust through the opening adjacent to the esophagus.

With such a poorly defined abnormality the progressive pathology like-

wise becomes a rather fortuitous process. A sufficient number of instances of diaphragmatic hernia are discovered routinely to suggest that the condition may frequently be arrested after development with a new pressure equilibrium established and give no difficulty. However such a course may not be presumed since gradual encroachment into the thoracic cavity and enlargement of the hernial opening is common. Where herniation is massive dysfunction of the heart, lungs and other thoracic organs may result as well as dysfunction of the displaced abdominal organs. Strangulation of the large or small intestine is a common sequel. The herniated section of the stomach may be pocketed and become infected. There seems to be a tendency to ulceration or malignancy of the stomach at the place where the herniated esophageal hiatus compresses the stomach wall.

There has been much rather fruitless definition of diaphragmatic hernias from a diagnostic point of view at least the distinction between true hernias with an enclosing sac and false hernias without such a sac is a blind alley. The presence or absence of such a sac is now believed to mean little etiologically and to have no definable effect upon the course of the hernia.

Clinical Symptoms — The greater part of the stomach may lie in thoracic

pulmonary hemorrhage may result from lung displacement and cyanosis may be noted particularly in children. Cardiac displacement may produce extreme pain resembling angina pectoris or coronary thrombosis. If the vagus nerves which pass in to the abdomen adjacent to the esophagus are constricted symptoms may be intense and various. A not infrequent finding is a severe and radiating pain running toward the left shoulder.

Physical Signs — The amount of information to be elicited by physical examination depends largely on the degree of displacement. If such displacement is small the degree of displacement. If such displacement is of the affected side is limited. If a gas-containing body such as the stomach lies in the thorax tympany will be noticed.

Roentgen ray Findings — Particularly when the hernia is a small one through the esophageal hiatus it may be overlooked in a superficial fluoroscopic examination. Diaphragmatic hernia is most readily detected with the patient supine. Lateral views are particularly helpful in the case of hernia through the esophageal hiatus. A complete examination requires a barium enema at the same time as the barium meal. With careful and

diaphragmatic hernia unless there is strangulation. In that case the symptoms are similar to those of infection—high sedimentation rate, etc.

Differential Diagnosis — In making a differential diagnosis one must consider emphysema and pyopneumothorax, congenital cysts and acquired

emphysema as evidence of the diaphragm and coronary thrombosis.

Imijje and Iyogun's observation—With these conditions a history of pneumonia or other lung infection can usually be elicited. They are usually accompanied by fever and other signs of toxemia. Although instances have been reported in which a hernia was thought to be emphysema and the



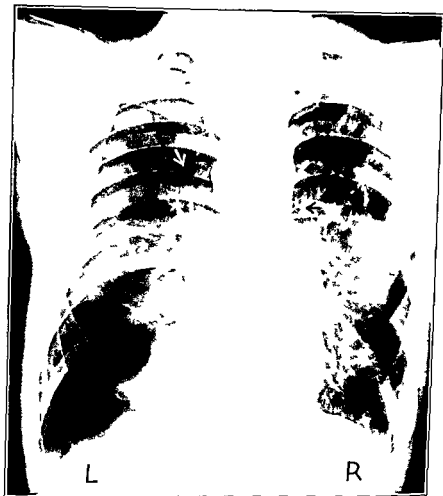
FIG. 18. Localized left lateral emphysema. I. P. female aged 35. Even a small portion of the air flow through the right lung is sufficient to cause this. These signs were well defined after 24 hours.

Diagnosis: Hemorrhage.

with hernia in the left

Both left and right diaphragmatic hernia and a large cyst may present a large, radiating area in

the thorax. Cyanosis is much more common with the latter condition and dyspnea is more likely to be severe. Cystic walls are thinner and more irregular as seen in a roentgen ray film than herniated stomach or intestine. Again a roentgen ray examination with barium is diagnostic.



Eventration of the Diaphragm —Where displacement of abdominal organs is so massive as to suggest either hernia or eventration, presence or absence of symptoms is almost diagnostic. With barium the location of the diaphragmatic shadow can be ascertained for diagnosis. (See Figure C, page 129.)

Coronary Thrombosis —The acute pain sometimes present in diaphragmatic hernia often suggests coronary thrombosis. Differentiation can

easily be made, however, on the basis of an electrocardiogram and roentgen-ray studies

BIBLIOGRAPHY

- BROWN S Diaphragmatic hernia, x ray diagnosis with 10 case reports, J Med 11, 520, 1930
- BROWN, S, and FINE, A Acute traumatic diaphragmatic hernia, 3 case reports, J Med 17, 443, 1936
- Diaphragmatic hernia its varieties and surgical treatment of hiatus type, Am J Surg 32, 204, 1936
- VINSON P P and MOERSON, H J Para-esophageal diaphragmatic hernia associated with intrinsic lesions in the esophagus report of cases, Arch Otolaryng 21, 437 1933
- WEINBERG, J Diaphragmatic hernia in infants, surgical treatment with use of renal fascia Surgery 3, 78, 1934

21. SUBPHRENIC ABSCESS

ALTHOUGH a subphrenic abscess is almost always a sequel to abdominal and not thoracic infection and although it is in itself entirely extra thoracic it frequently must enter into consideration when a chest diagnostician makes a differential diagnosis. Further, if untreated it may penetrate the diaphragm and even erode into a major bronchus. It is not a condition to be ignored in a discussion of thoracic diagnosis.

Etiology — Although subphrenic abscess may result from trauma penetrating wounds of the abdomen and occasionally a thoracic condition such as empyema or tuberculosis it follows an abdominal infection in the great majority of instances especially after abdominal surgery. Spread from the initial infection may be by lymph channels or blood stream occasionally as seen when the subphrenic abscess follows empyema or tuberculosis but in most instances spread is direct through the spaces of the abdomen. Infection is particularly likely to follow the gutter to the right of the ascending colon.

The abscess tends to confine itself to one of the six fairly well-defined areas defined by Barnard — the right and left extraperitoneal on the posterior portion of the liver where it is not covered by the diaphragm and the right and left posterior and anterior spaces. Location is in terms of the liver and the abscess tends to remain within the original areas defined by the triangular lateral and falciform ligaments.

infection next then gall bladder spleen and duodenum any abdominal infection can lead to a subphrenic abscess if neglected

The great majority of abscesses are found around the right lobe of the liver anterior and posterior. Stomach ulceration cancerous or not and infections of the spleen produce a somewhat smaller percentage of abscesses in the left anterior space. The extra peritoneal spaces are not usual sites.

Pathology — As in other abscess formations the subphrenic abscess begins as an area of intense inflammation with leukocytes moving out of engorged blood vessels toward the invading bacteria. These leukocytes collect around the bacteria. Necrosis of tissue and leukocytes gradually form a more or less spherical mass of liquid which is the abscess. In subphrenic abscess the bacteria are often of mixed character and the area and the infection are an important

factor in this walling-off. The patient may tolerate the abscess well for a time with only moderate symptoms. However the further process of healing with complete destruction of the bacteria by leukocytes and removal of the necrotic material seldom takes place without medical or surgical intervention. Although the abscess may be checked and seem static for a time it will usually progress to septicemia or bacteremia if neglected. The development of multiple abscesses is common and on

occasion the diaphragm is penetrated, with the formation of empyema pockets or erosion into lung and bronchus.

In addition to the usual general reactions to an infection focus there is almost always found concurrently a serous pleural effusion which may become infected about the abscess site. Contiguous abdominal organs may become inflamed but lymphatic spread when found is upward.

Clinical Symptoms—In most instances the initial symptoms of subphrenic abscess are masked by the primary infection. The first indication is usually prolongation of the symptoms of sepsis—fever and chills, malaise, headache, etc. At times, however, the onset may be abrupt with high fever, local pain, etc., interrupting normal recovery from the primary infection. In either case the development may suggest post-operative pneumonia.

ab
th
an

cess site is not usual but does occur.

Physical Signs The most consistent finding is the suggestion of impaired diaphragmatic movement on the affected side. There may be unilateral limitations of chest motion observable. In almost all instances some tenderness to percussion of overlying ribs can be elicited. A pleural friction rub or impaired resonance of the lower lung field due to serous effusion is usually detectable.

There is no consistent finding due to the abscess itself. If there is gas present in the cavity an abnormal area of resonance may be noted; otherwise it may be suggested merely by an unusually large area of liver dullness. Sometimes, particularly anteriorly, the abscess can be located by palpation; posteriorly located, some bulging of the flank may be seen.

Consistent or not, this is a fairly lengthy list of potential symptoms. The length of the list should not lull the diagnostician into less than eternal alertness for the possibility of subphrenic abscess; the percentage of late diagnoses of the condition is still quite high.

Roentgen-ray Findings The most consistent indication of subphrenic abscess is *immobilization or greatly impaired movement of the diaphragm on the affected (usually the right) side*. If the abscess is on the right side the diaphragm will be pushed upward somewhat but not with the usual smooth curve. On the left side it tends to be somewhat flattened. Obliteration of the costo-phrenic angle due to effusion is practically always found with obliteration of the cardio-phrenic angle if the precedent infection is of the liver.

Even when the area is obscured by inflammation both above and below the diaphragm the increased density of the abscess may be detected. Positive diagnosis by fluoroscope is more likely, however, where gas and a consequent fluid level is present.

Lateral views are vital in the examination for subphrenic abscess, since posteriorly located abscesses may lie completely in the diaphragmatic shadow in the anterior-posterior plane. Although the upright position is most informative generally, there is distinct advantage to varying the patient's

position before the screen, since other angles may show fluid level more clearly.

Laboratory Findings—The blood picture in subphrenic abscess is that characteristic of sepsis and is not specific. Bacteriological tests of the necrotic material of the abscess may be necessary where the antecedent infection is not clearly indicated by the patient's history.

A diagnostic aspiration for subphrenic abscess is never justified. The chief physiological defense of the body against general peritonitis is the

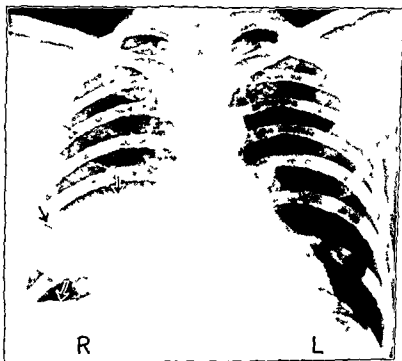


FIG. 70.—F. S. age thirty-four white male.

Incision and drainage of a subdiaphragmatic abscess was done on December 2, 1912 with aspiration of 1500 cc. thick purulent material.

Diagnosis: Right subdiaphragmatic abscess.

dense adhesions which rapidly wall off such abscesses, and the physician is never justified in penetrating this defense for mere diagnosis unless he is prepared to treat the abscess site at the same time. An exploratory

cavity with a serous exudate in this location it is a strong indication for a

diagnosis of subphrenic abscess. If empyema is found in this location aspiration of the pus followed by the injection of iodized oil may show it to be secondary to a connected subphrenic abscess.

Differential Diagnosis.

possibility of subphrenic abscess to be ruled out since occasionally such abscesses begin with blood borne bacteria localized by minor trauma or for reasons that remain undetermined. In such instances empyema lung abscess and pyopneumothorax echinococcus cyst of the liver and pneumonia must be considered.

Empyema Pyopneumothorax and Lung Abscess—These conditions usually present a history of pneumonia which however must be suspiciously regarded unless competently substantiated. Like subphrenic abscess they are characterized by continued or recurrent toxic symptoms. While the physical findings in the examination of the chest are apt to be more extensive than those of secondary effusion due to subphrenic abscess diagnosis cannot be made upon this basis. In most instances whether neither condition is clearly indicated by the patient's history, or by roentgenologic visualization of cavity diagnostic aspiration of the pleural fluid with the patient's chest lowered followed by the injection of iodized oil if the aspirated fluid is pus is necessary to diagnosis. After these techniques fluoroscopic study of the diaphragmatic area with the patient in various positions can establish the presence or absence of a pocket below the diaphragm. (See Figures 40 and 60 pages 72 and 117.)

Echinococcus Cyst of the Liver—This cyst will not present the marked sepsis of subphrenic abscess unless it has ruptured and become infected. The diaphragm will not be immobilized unless rupture has taken place. Differentiation between a ruptured echinococcus and a subphrenic abscess may be difficult if history or anaphylactic reaction is not informative in either case operation in the suspected area is in order.

Pneumonia—The pneumonia like the first group mentioned may be difficult to differentiate from subphrenic abscess on the basis of clinical symptoms. Where the patient's history is not suggestive differentiation can be made on the basis of roentgenologic study and physical examination. Strict localization of the chest shadows is diagnostic of lobar pneumonia in the other forms of pneumonia there will usually be suggestive shadows in the upper part of the lung or the opposite lung. Respiratory distress due to congestion—a characteristic of pneumonia—is usually lacking in subphrenic abscess. (See Figure 94 page 158.)

For other differential pictures refer to the appropriate chapters.

BIBLIOGRAPHY

- BOYCE, J. A. Pulmonary complications as aid in diagnosis of subphrenic abscess (presentation of case).
 BREWER, A. C.
 CARTER, H. N.
 35, 833 (1937)

- 1909, 1909, 1930
 OGDEN, A. and COOPER, A. M. & Lohs - 1930, 372, collected and
 England J Med
 n Surg 105, 496,
 STEPHENS, H. B. and ROUFF, I. Subphrenic abscess, Calif & West Med 40, 224
 1934
 THOREL, P. Six subphrenic spaces, applied anatomy and surgical considerations
 Surgery 21, 739 1947
 TRUESDALE, P. F. Origin and course of infection in subphrenic abscess, Ann Surg 98,
 846, 1933

THE TRACHEO-BRONCHIAL TREE AND THE LUNGS

22. ANATOMY AND MISCELLANEOUS DISEASES*

GROSSLY the respiratory organs are seen as two sacs completely enclosed in the pulmonary or parietal pleura with the 4 or 5 inch tube of the trachea lying between the upper half of them. Each lung is enclosed in its own separate parietal pleura which is continuous with the visceral pleura in the fold joined to the lung at the hilus. When a cut is made

in the pleural cavity. It is normally connected to the other structures only by the juncture of the trachea with the larynx and by the blood vessels. The visceral pleura connected to the lung by the triangular fold described the pulmonary ligament is in turn connected to the first rib and the diaphragm.

The apices of the lungs extend from 1 to 2 inches above the level of the first rib in a sort of rounded dome. The right lung is usually slightly higher than the left and since it is also shorter though wider and heavier than the left the base of the right lung is visibly higher. Both lungs tend to be somewhat conical in shape with their inner edges roughly perpendicular. The base of the left lung lies at about the level of the sixth chondrosternal articulation and slopes down approximately paralleling the sixth rib. The base of the right lung slopes at about the same degree but as noted is somewhat higher. Both bases are slightly concave. The outer surfaces of the lungs are rather regularly rounded except for the indentation of the ribs. The mediastinal surfaces are quite irregular with indentations for the pericardium and the large vessels in this area.

The visceral pleura is in function a skin around the lung and is as closely associated with it as skin is with other body tissues. This pleura extends deeply into the lung in the interlobar fissures. The lung itself is divided into secondary lobules quite clearly distinguished when the pleura is removed by connective tissue especially rich in lymphatics. The lobules correspond in some degree to bronchial branching although Brock demonstrates that the relation of bronchi to lung division is by no means uniform.

The trachea, bronchi, bronchioles and alveolar tissue with blood vessels, lymphatics and connective tissue form a single functional unit. The trachea is a cylindrical tube slightly flattened posteriorly extending from the cricoid cartilage of the larynx to its bifurcation into the right and left

bronchi. The bronchi themselves extend deep within the lung branching extensively. They are more truly cylindrical than the trachea and each section from one branching to the next is of uniform diameter. The bronchi terminate in bronchioles distinguished grossly from the bronchi by the appearance of occasional alveoli along their walls. (The further distinction of middle-sized air passages seems pointless.) Bronchioles terminate in turn in alveolar ducts with atria which lead to alveolar sacs where the function of respiration is largely accomplished.

The trachea is formed around a framework of irregularly shaped and spaced rings of hyaline cartilage. These cartilage rings are incomplete and somewhat flattened posteriorly. They are connected by bands of transverse smooth muscle. Longitudinal bands of elastic fibers lie on top of the cartilage and below the smooth muscle. Unless age or abnormality has made the cartilage fibrous, this arrangement makes for a surprising degree of flexibility. Externally the tracheal cartilage is sheathed in connective tissue and the interior of the tube is lined with tunica mucosa lying on a layer of loose connective tissue. The tunica mucosa the epithelium lining the trachea is composed of four types of cells: basal, intermediate, columnar ciliated and goblet. This epithelium rests on a basement membrane composed of reticulum with occasional collagenous fibers. The four cell types are actually stages of development from the basal undifferentiated cell. The columnar ciliated cell is the mature form, the

verse smooth muscles

In an adult the bifurcation of the trachea into the two bronchi ordinarily takes place about the level of the seventh vertebra. The right bronchus can be viewed as an extension of the trachea with the left as an offshoot since the former departs only about 20 degrees from the perpendicular of the trachea and the latter swings off at least twice that angle.

than the diameter
near the bronchi very
lymphatic onch and
trachea but as they progress further into the lung the cartilage becomes
more irregular and with of
crease in the percent if
thickness decreases 1
by cuboidal forms

Anatomically the most useful distinction between bronchi and bron-

as marking the change from bronchus to bronchiole. There is no concurrent abrupt change in construction, but a gradual change is noted as tube diameter diminishes. The substitution of cuboidal ciliated cells for columnar becomes more frequent with the columnar cells finally disappearing entirely. In the bronchioles basal cells are found only infrequently. A further modification takes place when the cuboidal cells become non-ciliated. The final modification is the replacement of the ciliated columnar and cuboidal cells by stratified squamous epithelium. This type of epithelium can be found in the bronchioles, but is more commonly found in the terminal bronchioles. The basement membrane persists throughout the bronchioles but becomes thinner with the diminishing tube diameter.

secondary in function to the basic unit of the lung the primary lobule. This lobule begins at the termination of the respiratory bronchiole. ordinarily several lobules to each termination. It consists of an alveolar duct which terminates in several atria which in turn lead to two or more alveolar sacs.

The enclosing element in the walls of the alveolar sacs, as well as bronchiole alveoli is elastic fiber. This fiber has been mentioned as lying in bunches in the trachea; it is continuous throughout the tracheo-bronchial tree. In the bronchi and bronchioles it lies irregularly but generally horizontally, connecting with the intersecting smooth muscles. When it reaches the alveoli and alveolar sacs it first encircles the root of the sac and then forms

as a modification of the epithelial cells of the lining

to the wave-like motion of the cilia and the peristaltic motion of the bronchi initiated by the horizontal smooth muscles are effective in removing foreign matter by moving it toward the trachea. In the trachea these two mechanisms continue to operate aided by the cough reflex. The inefficiency of any of these mechanisms in a disease process may seriously impede restorative processes. Post-operative atelectasis due to a block

ing of a major bronchus may be due in some measure to such inefficiency.

The tissue of the lungs is fed by the bronchial arteries. These may vary in number although there is ordinarily one such artery for the right lung and two for the left. The left bronchial arteries arise from the thoracic aorta the right artery is frequently an offshoot of one of the left. These arteries run along the back side of the bronchi within the surrounding connective tissue. They are inter connected by anastomotic branches.

not beyond the third division of the bronchial tree. They empty into the azygos vein.

The pulmonary artery arises from the right ventricle of the heart. It extends upward and backward until it lies behind the ascending aorta but in front of the tracheal bifurcation. At this point the pulmonary artery also divides and enters the lung in front of and slightly below the bronchus. Thereafter the two are consistently adjacent with the artery sometimes lying within the connective tissue of the bronchus. The pulmonary artery follows all bronchial branching to each primary lobule. About the beginning of the respiratory bronchiole the artery puts forth branches which form a comparatively coarse network about the bronchiole and succeeding structures and nourishes them. The main branches continue to each individual lobule where they terminate in a very fine and dense capillary network completely surrounding the air sacs. Here the oxygenation of the blood is accomplished.

The newly oxygenated blood from the alveolar capillaries is only one source of the blood returning to the heart by the pulmonary veins. These

and venous blood to be oxygenated are by no means distinct entities. Unlike the arteries the pulmonary veins return to the hilus independently and at a distance from the bronchial passages. These veins gradually unite until there are just two that pass from each lung (the veins of the middle lobe of the right lung joining those from the upper) and enter the left atrium of the heart. Unlike other pulmonary blood vessels the pulmonary veins are not valved.

The lungs also contain two of the most extensive networks of lymphatics in the body. The deep lymphatics follow, in an irregular and tortuous network the pulmonary veins, arteries and bronchi as well as forming a very close web in the connective tissue between the secondary lobules. The superficial lymphatics are found within the pleura. Throughout both systems unidirectional flow of lymph is maintained by a system of valves. These valves are so situated that lymph from the lung may find its way into the pleural system but not the other way around. Both systems empty into the clusters of lymph nodes around the hilus. Lymph nodes are also found within the lung particularly at bronchial arterial branchings. Also found

scattered rather haphazardly throughout the lymphatic system are patches of unorganized lymphatic tissue.

The lymphatic system is not like that of the blood, a closed system. The irregular branching lymph channels terminate in a capillary system characteristically arranged in a spiral or ring-like pattern, which tends to be thought of as a one-way valve.

It is by working between tissue cells. When there is no interference with the system, these tissue fluids enter the lymph channels at a rate sufficient to provide a steady, if sluggish, stream of lymph fluid, emptying eventually into the thoracic duct and then into the venous blood. The chief pathological

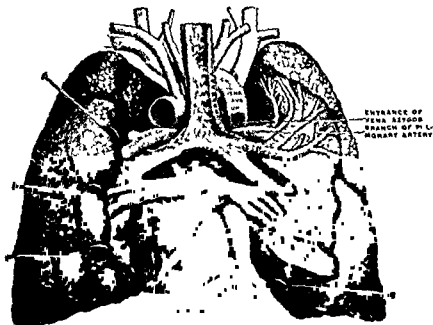


FIG. 71. Dorsal view of the heart and lungs. The lungs have been pulled away from the median line, and a part of the right lung has been cut away to display the air-ducts and blood-vessels. (Costut.)

significance of the lymphatics is that they provide a means of removal of foreign matter. Such matter is usually phagocytosed and carried into the system also intracellularly. In the case of bacteria and malignancy, the lymph system is a route of proliferation.

The nerve supply of the lungs is from two sources, vagus nerves arising in the medulla oblongata and a sympathetic system arising from the second, third and fourth ganglia of the spine. Both systems contain both motor and sensory nerves. The systems unite to form plexi posterior and anterior at the root of the lung. Fibers are given off from these plexi that join the main bronchi and arteries at the hilus. The chief afferent nerves of the bronchi

ing of a major bronchus may be due in some measure to such inefficiency.

The tissue of the lungs is fed by the bronchial arteries. These may vary in number although there is ordinarily one such artery for the right lung and two for the left. The left bronchial arteries arise from the thoracic aorta; the right artery is frequently an offshoot of one of the left. These arteries run along the back side of the bronchi within the surrounding connective tissue. They are inter-connected by anastomotic branches to the terminal arteries. In

The bronchial veins do not extend very deeply into the lung ordinarily not beyond the third division of the bronchial tree. They empty into the azygos vein.

The pulmonary artery arises from the right ventricle of the heart. It extends upward and backward until it lies behind the ascending aorta but in front of the tracheal bifurcation. At this point the pulmonary artery also divides and enters the lung in front of and slightly below the bronchus. Thereafter the two are consistently adjacent with the artery sometimes

a comparatively coarse network about the bronchiole and succeeding structures and nourishes them. The main branches continue to each individual lobule where they terminate in a very fine and dense capillary network completely surrounding the air sacs. Here the oxygenation of the blood is accomplished.

The newly oxygenated blood from the alveolar capillaries is only one source of the blood returning to the heart by the pulmonary veins. These veins also carry the blood returning from the pleura; the alveolar ducts and

at a distance from the bronchial passages. These veins gradually unite until there are just two that pass from each lung (the veins of the middle lobe of the right lung joining those from the upper) and enter the left atrium of the heart. Unlike other pulmonary blood vessels, the pulmonary veins are not valved.

The
in the
work
close web in the connective tissue between the secondary lobules. The superficial lymphatics are found within the pleura. Throughout both systems unidirectional flow of lymph is maintained by a system of valves. These valves are so situated that lymph from the lung may find its way into the pleural system but not the other way around. Both systems empty into the clusters of lymph nodes around the hilus. Lymph nodes are also found within the lung particularly at bronchial arterial branchings. Also found

scattered rather haphazardly throughout the lymphatic system are patches of unorganized lymphatic tissue

The lymphatic system is not like that of the blood, a closed system. The

working between tissue cells. When there is no interference with the system these tissue fluids enter the lymph channels at a rate sufficient to provide a steady if sluggish, stream of lymph fluid emptying eventually into the thoracic duct and then into the venous blood. The chief pathologic

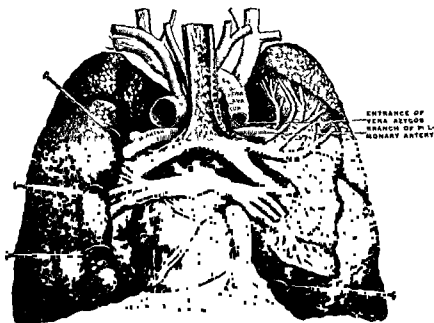


FIG. 71.—Dorsal view of the heart and lungs. The lungs have been pulled away from the median line and a part of the right lung has been cut away to display the air-ducts and bloodvessels. (Testut.)

significance of the lymphatics is that they provide a means of removal of foreign matter. Such matter is usually phagocytosed and carried into the system also intracellularly. In the case of bacteria and malignancy the

third and fourth ganglia of the spine. Both systems contain both motor and sensory nerves.

the root of the lung
bronchi and arte

ing of a major bronchus may be due in some measure to such inefficiency.

The tissue of the lungs is fed by the bronchial arteries. These may vary in number although there is ordinarily one such artery for the right lung and two for the left. The left bronchial arteries arise from the thoracic aorta the right artery is frequently an offshoot of one of the left. These arteries run along the back side of the bronchi within the surrounding connective tissue. They are inter-connected by anastomotic branches. The bronchial arteries supply all the tissue of the lungs except the terminal bronchioles and alveoli these are supplied by the pulmonary arteries. In man branches of the bronchial artery also supply the pleura.

The bronchial veins do not extend very deeply into the lung ordinarily not beyond the third division of the bronchial tree. They empty into the azygos vein.

The pulmonary artery arises from the right ventricle of the heart. It

lies within the connective tissue of the bronchus. The pulmonary artery follows all bronchial branching to each primary lobule. About the beginning of the respiratory bronchiole the artery puts forth branches which form a comparatively coarse network about the bronchiole and succeeding structures and nourishes them. The main branches continue to each individual lobule where they terminate in a very fine and dense capillary network completely surrounding the air sacs. Here the oxygenation of the blood is accomplished.

The newly oxygenated blood from the alveolar capillaries is only one source of the blood returning to the heart by the pulmonary veins. These veins also carry the blood returning from the pleura the alveolar ducts and atrium and the blood from the capillaries of the bronchial artery beyond the limits of the bronchial veins. Thus the system of blood supply to the lungs and venous blood to be oxygenated are by no means distinct entities. Unlike the arteries the pulmonary veins return to the hilus independently and at a distance from the bronchial passages. These veins gradually unite until there are just two that pass from each lung (the veins of the middle lobe of the right lung joining those from the upper) and enter the left atrium of the heart. Unlike other pulmonary blood vessels the pulmonary veins are not valved.

The lungs also contain two of the most extensive networks of lymphatics in the body. The deep lymphatics follow in an irregular and tortuous network the pulmonary veins arteries and bronchi as well as forming a very close web in the connective tissue between the secondary lobules. The superficial lymphatics are found within the pleura. Throughout both systems unidirectional flow of lymph is maintained by a system of valves. These valves are so situated that lymph from the lung may find its way into the pleural system but not the other way around. Both systems empty into the clusters of lymph nodes around the hilus. Lymph nodes are also found within the lung particularly at bronchial arterial branchings. Also found

scattered rather haphazardly throughout the lymphatic system are patches of unorganized lymphatic tissue.

The lymphatic system is not like that of the blood, a closed system. The irregular branching lymph channels terminate in a capillary system characterized by knobby rather club-like endings. No regular openings to these terminations have been satisfactorily demonstrated and it is thought that the tissue fluids which compose the lymph enter the channels by working between tissue cells. When there is no interference with the system these tissue fluids enter the lymph channels at a rate sufficient to provide a steady if sluggish stream of lymph fluid emptying eventually into the thoracic duct and then into the venous blood. The chief pathologic

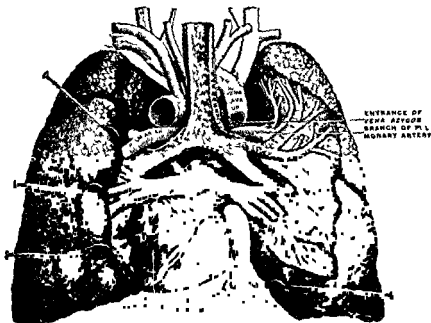


FIG. 71. Dorsal view of the heart and lungs. The lungs have been pulled away from the mediastinum and a part of the right lung has been cut away to display the air-ducts and blood-vessels. (Testut.)

significance of the lymphatics is that they provide a means of removal of foreign matter. Such matter is usually phagocytosed and carried into the system also intracellularly. In the case of bacteria and malignancy the lymph system is a route of proliferation.

The nerve supply of the lungs is from two sources, vagus nerves arising in the medulla oblongata and a sympathetic system arising from the second

arise in the epithelium of the atria and around bronchial branchings and in the smooth muscle. The efferent nerves terminate in the bands of smooth muscle and the muscular sphincters around the alveoli. The nerves of the arteries terminate in the *tunica media*.

The impulses effecting respiration originate, within the lung, largely at the receptors in the alveolar atria, and without the lung in receptors in the carotid artery and aortic arch. The atrial impulses are caused by the distension of the walls upon inspiration, the receptors within the arteries are chemical responding to changes in carbon dioxide and oxygen content.

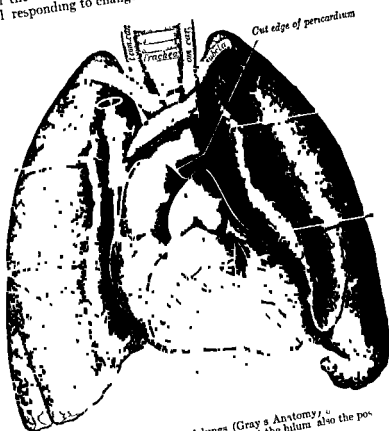


Fig. 72.—Anterior view of heart and lungs (Gray's Anatomy), showing lobes of the lungs with the associated blood vessels at the hilum, also the position of the heart.

The chief efferent nerve in respiration is the phrenic nerve, controlling diaphragmatic movement, but subsidiary muscles to the chest wall and subsidiary nerves come into play when the diaphragm is paralyzed. Inspiration is the active phase of respiration, but expansion of the lungs is indirectly accomplished by the negative pressure gradient created by enlargement of the chest cavity.

The foregoing summary of the anatomy of the lung is obviously not offered as a complete description of lung anatomy but as a survey prior

to the investigation into the various pathologic conditions commonly

but for a more detailed description the reader is referred to strictly anatomical studies particularly those of Brock and Miller and of course Gray

Miscellaneous Lung Diseases — There are several lung conditions which are important to differential diagnosis but which do not merit prolonged examination either because they are infrequently encountered or because

examination. Its manifestations are quite various. A partial stenosis will

emphysema with mediastinal displacement and pronounced thoracic asymmetry is the result. The effect of complete occlusion will of course depend upon the location. Atelectasis will occur only if the stenosis is of a major bronchial branch prohibiting collateral circulation of air through alveolar walls.

Diagnosis of bronchial stenosis is comparatively simple once it is suspected and bronchoscopy and a roentgen ray examination is undertaken. A roentgen ray examination with a contrast medium is very informative since if the occlusion is not complete some oil will seep through the stenotic area and outline it showing its extent.

As indicated the discovery of stenosis is preliminary the diagnostic question of most importance is the cause of the condition. Bronchoscopy is the most useful weapon for this diagnosis in most instances. The most frequently encountered intrinsic causes are new growths, edema from the inhalation of irritating fumes, irritation by foreign bodies and injuries and wounds and resulting scar tissue or adhesions. Stenosis due to bacterial lesions such as tertiary syphilis or tuberculosis is increasingly rare. Bronchial stenosis can also be produced externally by enlarged goiters, aneurysms, enlarged or infected glands or lymph nodes or new growths. However such extrinsic stenosis is not common. Space-taking growths in the mediastinum are much more likely to affect the patient by compression of blood vessels than by compression of the trachea or bronchi. (See Figure

now is suspected of course when a child develops a respiratory difficulty. It is usually uncorrected to helpful but failure to elicit such an incident does not preclude foreign bodies. They

are able to remain quiescent for long periods, and thus be forgotten or they are frequently aspirated during unconscious states.

Physical signs and symptoms will depend upon the size, location and adjacent tissue changes. Where aspiration is followed by an immediate reaction due to the foreign body itself the chief symptoms are pain and dyspnea. Spasmodic cough is also a feature. Dullness to percussion will be noted if a bronchus is occluded to produce atelectasis. Diagnosis will be most difficult however if symptoms are inflammatory, the patient's history is less informative. Such changes are inflammatory, the patient may have pronounced fever and malaise and cough up pus. These tissue changes frequently occlude the bronchus around the foreign body and produce atelectasis.

Where the foreign body is opaque diagnosis is made readily by roentgen-ray examination. Pictures from several angles should be made to prevent the shadow of the foreign body being concealed in the mediastinal shadow and for exact localization. This is particularly necessary when a large body in the trachea is suspected and dyspnea is a pronounced symptom, such as dyspnea is frequently the result of the lodgment of the foreign body in the esophagus with extrinsic stricture of the trachea or spasmodic reflex producing the dyspnea. Where the roentgen ray examination does not demonstrate a foreign body, bronchoscopy will almost always reveal the nature of the abnormality. Where access shadows lie deep in the parenchyma bronchoscopy under fluoroscopic control will frequently enable the examiner to determine the nature of the abscess. (See Figure 21, page 57.)

Broncholithiasis is defined as a condition in which calculi are formed in the bronchi. The term covers a multiplicity of etiologically distinct conditions. The distinction under such circumstances between broncholithiasis and pulmonary calcification seems rather pointless to diagnosis since a pulmonary calcification can at any time break loose from its site of deposition or penetrate a bronchus if it has been deposited in the lymphatic system and become indistinguishable from a broncholith. So far as applied to those instances in which the primary predisposition is in the lung itself medicine is concerned the most useful division is in the lung it self into those instances in which the primary predisposition is in the lung result from a disorder and those in which deposits of calcification in other parts of the body in blood composition due to abnormality in other parts of the body.

The latter group in which lung stones represent a reaction to a dysfunction seated elsewhere is not common but must be kept in mind during the examination. Of course kidney and glandular dysfunction is the commonest cause of calcium deposit. If kept in mind this origin is seldom misleading since there will usually also be symptoms referable to those organs. Important but still rarer are lung stones due to bone tumor. In any instance of broncholithiasis a thorough physical examination is in order. The commoner cause of broncholithiasis is local infection. Individual metabolism and blood composition are of course predisposing factors but need not be outside normal limits. In this group tuberculosis and histoplasmosis and other fungi are apparently the chief etiologic agents. Such calcification may be scattered widely throughout the lungs representing healed foci or may be clustered around the tracheal bifurcation or a hilus. In the latter instance the calcification is of lymph nodes. The acute stage

of the infection may be without memorable symptoms. Occasionally local bronchitis or a foreign body may cause local calcification. Bronchiectasis is stone producing at times. In general it may be said that any necrotic or chronically irritated tissue of the bronchi or parenchyma may at times occasion the deposit of calcium.

It seems probable that in all but exceptional instances the lung makes some attempt to fibrose any rough stone within the bronchi or parenchyma. Particularly in the case of stones of tuberculous or fungus origin this fibrosis is frequently effective. As long as the deposit remains in one place particularly if it is fibred the patient may remain symptomless. There is

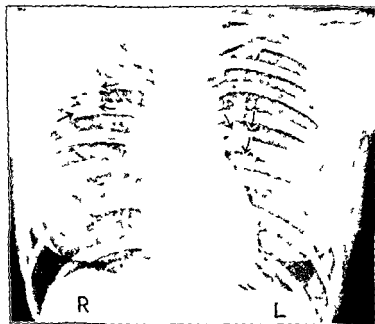


FIG. 73. D. P. age twenty-six with history of
Admitted to the hospital several years between September 2, 1944 and present.

tendency, however, for calcification to produce symptoms after years of quiescence. Riley found half of his patients with tuberculous calcifications to have symptoms.

The symptoms of bronchiectasis itself are mechanical, produced by blocking and irritation. After a stone has been dislodged into the air passages it is in effect a foreign body. The defensive mechanisms of the

lung tend to move it into the trachea for expectoration. Like a foreign body the symptoms are various. Stenosis and atelectasis may occur. Dyspnea and paroxysmal cough are common. The irritation usually results in inflammation with much sputum and some degree of hemoptysis and abscess beyond the dislodged stone or bronchiectasis, may be a sequel. Since such stones are in composition usually very much like bone it follows that they are readily visible by roentgen ray. However, much difficulty may result from stones too small to be seen readily, and lateral and oblique views are necessary to demonstrate stones in the superimposed heart and mediastinal shadow. The distinction between calcified nodes and broncholiths within the bronchi may be difficult. Bronchoscopy may help in diagnosis and where abscess is present exploration and open drainage is a reasonable procedure. In all instances of troublesome lung stones the possibility of an active underlying pathology particularly hyperparathyroidism or renal tumor should be eliminated by a thorough examination.

BIBLIOGRAPHY

ANDERSON W S and MACKAY J B Broncholithiasis. *Dis of Chest* 10 427 1944
BLOOM W Histogenesis of essential lipoid histiocytosis. *Arch Path* 6 827 1928
BROCK R C The Anatomy of the Bronchial Tree. New York Oxford University Press 1946
BROWN A I Foreign body in the esophagus for three years. *Calif & West Med* 34 371 1931
COUNCILMAN W T The lobule of the lung and its relations to the lymphatics. *J Boston Soc M Sc* 4 1900
FLOESER I and FREEMAN A Model to illustrate mechanics of respiration and movements of mediastinum with and without various kinds and degrees of bronchial stenosis. *Surgery* 3 774 1938
FRIED B M Origin of histocytes (macrophages) in lungs. experimental study by use of intratracheal injections of vital stain. *Arch Path & Lab Med* 3 751 1927
GARDNER I I Studies on relation of mineral dusts to tuberculosis. I relatively early lesions in experimental pneumokoniosis produced by granite inhalation and their influence on pulmonary tuberculosis. *Am Rev Tuberc* 4 734 1920
GRAHAM F A SINGER J J and BALLON M C Surgical Diseases of the Chest. Pl 11. *Philadelphia Lea & Febiger* 1935 pages 502 507 525 535 562 & 564
GRAY H Anatomy of the Human Body. 20th Ed. Philadelphia Lea & Febiger 1948 p 1129
GRETHMANN W Architecture of terminal sections of bronchi of human lung. *Am Rev Tuberc* 31 261 1935
HANSEN PRUSS O C and GOODMAN E G Allergic pulmonary consolidations. *Ann Surg* 42 266 1938
HEAD J R Surgical indications in perforation of esophagus by foreign bodies. *Am J Surg* 54 193 1941
HOLINGER P and ANDREWS A H Jr Bronchial obstruction signs symptoms and diagnosis. *Am J Surg* 54 193 1941
IGLAUER S Nonopaque foreign bodies in esophagus and esophagotracheal fistula. their demonstration by ingested iodized oil. *Arch Otol* 7 229 1928
JACKSON C and JACKSON C I Faulty surgical instruments as cause of foreign body. *Arch Otol* 22 293 1935
Staples and double-pointed tacks as foreign bodies. mechanical problems of bronchoscopic extraction. *Arch Otol* 22 603 1935
Diseases of the Nose Throat and Ear. Including Bronchoscopy and Esophagoscopy. Philadelphia W B Saunders 1945
JACKSON C I Endoscopy for foreign body report of 178 cases of foreign body in ear and food passages. *Ann Otol Rhin & Laryn* 45 644 1936
JOSELYN L I Nature of pulmonary alveolar lining. *Anat Rec* 62 147 1933
KOURILSKY R FOURESTIER D B and PROVO A Remarkable tolerance of three practically normal subjects to long standing unrecognized bronchial stenosis of tuberculous origin. *Rev de tuberc* 10 609 1946

- LEMON, W. S. Anatomic factors involved and mechanical devices employed in removal of minute particles of foreign material from lungs: experimental study. *Tr Assoc Am Physicians* 52: 278, 1937.
- MACELIN, C. C. Alveolar pores and their significance in human lung. *Arch Path* 21: 202, 1936.

Tuberc 52: 503, 1945.

- VAN ORDSTRAND, H. S., MOORE, M. Jr. and HARRIS, H. E. Broncholithiasis: report of 2 cases. *Cleveland Clin Quart* 9: 36, 1942.
- WEINBERG, J. Bronchial obstruction produced by organic and inorganic foreign bodies. *J Thorac Surg* 7: 498, 1938.
- WILLSON, H. G. Terminalis of human bronchiole. *Am J Anat* 30: 267, 1922.
- ZWEIFEL, E. Über zwei Fälle von Broncholithiasis. *Zur Differentialdiagnose der Bronchopneumonie*. *Schweiz med Wchschr* 74: 833, 1944.

23. BRONCHIECTASIS

BRONCHIECTASIS is a chronic usually progressive disease of the lower respiratory tract. Although ordinarily originating as a secondary condition it persists with its own characteristics after the elimination of the primary pathology. Anatomically it is characterized by dilatations of the bronchi or bronchioles or both. The degree of dilatation is in some degree a measure of the progress of the disease.

Etiology—Bronchiectasis can be either congenital or acquired. The former is caused by obstruction of the bronchi by bands of pleura during fetal life. Its progress is much less marked than the acquired type and it is ordinarily not discovered except by accident.

Acquired bronchiectasis ordinarily follows pneumonia, tuberculosis, malignancy or atelectasis from any cause. In all cases the etiological factor is damage to the bronchial walls resulting from infection or from inflammation of the peribronchial tissue. A very important cause of bronchiectasis is the lodgment of foreign bodies, particularly vegetable bodies in the bronchus. Not uncommonly a broncholith may occlude a large bronchus and lead to bronchial infection, abscess formation and eventually a bronchiectatic area. In childhood bronchiectasis is most commonly a complication following bronchopneumonia, particularly when the latter results from whooping cough or measles.

Bronchiectasis is a common complication of advanced silicosis and tuberculosis in the chronic form. In cases of stenosis of the smaller bronchi bronchiectasis usually develops beyond the stenosis. Tumors and cysts within the bronchial walls may block the lumen to such a degree that large areas of atelectasis develop. Secondary inflammation with suppurative bronchial dilatations may follow.

Chronic infection of the nasal sinuses is frequently coexistent with bronchiectasis and an acute infection of one may lead to similar infection of the other.

Pathology—In congenital bronchiectasis there is a more or less orderly arrangement of the columnar epithelium, cartilage plates and mucous glands. Inflammation is either non-existent or slight. Cylindrical enlargements of the bronchi with atelectasis of the surrounding lung tissue is the usual type of involvement. This characteristic persists when a primary pathology, such as an abscess formation due to pneumonic infection, might lead the examiner to suspect acquired acute bronchiectasis. Multiple lung cysts may be present. They are usually distributed throughout the entire lung but they are occasionally found confined to a single lobe when the main bronchus is compressed.

Acquired bronchiectasis because of its varied etiology cannot be pathologically defined with any strictness. The primary characteristic is a persistent dilatation of the bronchi as in congenital bronchiectasis. The enlargements are not however typically cylindrical although cylindrical enlargements may be present among saccular and fusiform ones. The disease is almost always encountered with episodes of acute infection.

A quiescent form seems to result in rare instances when the acute stage has been overcome with or without treatment. Although the bronchial tract is dry and normal the dilated passages still remain and the disease at least technically still exists. The instances reported by Blades in which typically dilated bronchi as seen with iodized oil roentgenograms subsided after a few weeks probably do not represent true bronchiectasis. It is conceivable that there were relaxations of the bronchial walls as a secondary complication to an inflammatory process (in most cases atypical pneumonia) which were not due to tissue changes and so returned to normal when the primary inflammation subsided. True bronchiectasis most probably involves a tissue degeneration.

In the acute stage of bronchiectasis the stage in which it is most often seen changes occur within the bronchial walls. Infection may begin in the mucosa and lead to considerable excretion of mucus and pus. The characteristic dilatation of the bronchial walls produces strictures and destructions of the bronchial tree above the dilated portion oftentimes such an area is suitable soil for putrefactive organisms which may cause abscess or even gangrene of the lung. The bronchial wall may be ulcerated and if a large blood vessel is eroded severe hemorrhages may take place. Metaplasia even keratinization may be present. The presence of fibrous strictures in cases of bronchiectasis is common. It is probable that these are produced by earlier infections of the tracheo-bronchial tree and then in turn produce bronchial dilations beyond the strictures. Pleural fibrosis may accompany bronchiectasis when that disease is complicated by secondary pneumonitis.

Clinical Symptoms.—Where bronchiectasis is suspected a history of pneumonia following chicken pox or whooping cough in childhood is important to diagnosis like wise the aspiration of a foreign body at any time by the patient should be investigated. A family history is worth while since there is an unexplained tendency for bronchiectasis to occur in two or more members of the same family. In this connection situs transversus and anomalous running in families is frequently associated with bronchiectasis. Coughing is the most important and persistent symptom. It may be a slight hacking cough with slight expectoration on the other hand it may be a violent pyrexia with large amounts of sputum. The history and physical will show that the cough has persisted for very usually from childhood. The expectorated material is commonly malodorous although not always. The odor in some cases is so offensive that the patient becomes an outcast. Hemorrhages are more frequent in advanced cases than in tuberculosis and occasionally fatal hemoptysis occurs. Very rarely a true bronchiectasis is encountered that is not marked by cough or the other symptoms normally present but only by lung hemorrhages. Bazançon has called it dry bronchiectasis. This form is most frequently mistaken for tuberculosis (see differential diagnosis section). Constitutional symptoms may be slight for long periods of time unless there is pneumonia or pleurisy. In attacks of bronchiectatic pneumonia often experience fever, sweats and chills. These symptoms occur with the attacks of pneumonia which fre-

quently develop as a result of the spread of the primary infection from the bronchi through the lymph channels to contiguous lung tissue

Osteoarthropathy is a common accompaniment to bronchiectasis and in long standing cases dyspnea is not an unusual symptom due to emphysema and cor pulmonale (See Figure 1 page 13)

Physical Signs — There are no physical signs truly typical of bronchiectasis. Rales over the area of the lesions is the most common sign but it is by no means universal. Sometimes impaired resonance of the area is noted. In patients who have large amounts of sputum retained in the bronchial dilatations diminished breath sounds are found if these same patients are given postural drainage or have a violent cough with much expectoration bronchial sounds will become distinct and whisper sounds will markedly increase due to the thickening or fibrosis of the area.

In patients who have bronchiectasis with atelectasis the compensatory emphysema which develops surrounds the affected lobe and signs otherwise elicited by auscultation and percussion are masked.

In patients who have had bronchiectasis for a long time considerable atelectasis may be present. Particularly if the disease is unilobular this atelectasis may be indicated by imperfect chest expansion. Likewise it is in this same type of situation that we may expect to find osteoarthropathy the clubbing of the fingers and toes.

Patients who at times show marked percussive and auscultative findings may at other times seem completely normal. The illusive nature of physical signs in bronchiectasis makes this disease very difficult to diagnose by physical examination alone. Such an examination can be helpful however where signs exist in locating the pathologic area and determining its approximate extent.

Roentgen ray Findings It is important to view patients fluoroscopically from various angles when bronchiectasis is suspected. The disease often occurs in the basal area of the tracheo bronchial tree where roentgenograms from the usual angles leave the affected area in the cardiac shadow. A lateral view will often reveal bronchiectatic atelectatic areas that are not visible in posterior anterior views.

With the ordinary roentgenogram it is possible to see the varying degrees of air containing lung and the enlarged and slightly denser bronchi. A displacement of the heart or mediastinum is significant and the movements of the diaphragm should be observed. Large cavities with fluid levels may be visible. Where cysts coexist with the bronchiectasis they may be seen as circular or oval areas of increased density. If the bronchiectatic area is complicated by pneumonitis it too may appear somewhat denser than normal tissue.

There need be present in bronchiectasis only indications that suggest the disease. However a conclusive

diagnosis can be made in almost all cases by the use of the roentgen ray in conjunction with a contrast medium. The author has found lipiodol consisting of 40 per cent by weight of iodine with poppy seed oil the most effective contrast medium. However a wide variety of preparations can

taining various types of oil and varying percentages of iodine or other contrast mediums are also used.

There are several methods of injecting the oil into the bronchus. The method described by the author is the use of a local anesthetic. The oil is injected into the trachea as far as possible. The injection may be made into that area (page 34) or into the solution sprayed into the

trachea or nasal catheters are sometimes used for this injection also but with this technique a local anesthetic is required.

The bronchoscope offers a very useful method of injecting iodized oil. It permits the oil to be injected directly into the bronchus under suspicion without affecting other areas of the lung. This permits the bronchus to be studied more effectively because there are no iodized oil shadows in the other areas to confuse the examiner. Such a study, however, must be made immediately after the injection since the oil tends to spill over from one bronchus to another. Whenever the bronchoscope must be used to aspirate material that interferes with the penetration of the iodized oil, the oil should be injected through a tube in the bronchoscope before the instrument is removed.

By the methods outlined above bronchiectasis can usually be definitely recognized. The bronchi of the infected area are seen to be dilated and distorted. They are also usually compressed into a smaller area due to the shrinkage that takes place in an affected lobe. The methods outlined above are of course immediately accurate with considerable accuracy. The relation between type and course or etiology has not been conclusively demonstrated.

Normally most of the iodized oil will be expelled in four to eight hours after its administration with expectoration starting almost immediately. It is important therefore that the roentgenograms be taken immediately after the injection. It was formerly thought that roentgenography several days after an injection gave further indication of diseased tissue since such roentgenograms indicated a wider area. It is now recognized that this results from iodized oil penetrating the finer healthy alveoli and that such roentgenograms actually distort the examiner's picture of the extent of the disease. This persistence is now recognized as a reason for spacing roentgenographic studies with iodized oil far enough apart for the condition to disappear.

There have been instances seen by the author in which the iodized oil has been retained in the alveolar structures for weeks. Ordinarily this has no ill-effects.

A few times oil has been injected into a portion of the lung sensitive to iodine, the discharge and congestion, slight fever and headache after a few days. It is not serious and the symptoms disappear.

Localized by nasal injection sometimes accompanied by a

BRONCHIECTASIS



FIG. 74 — Bronchiectasis. After iodized oil injection one notes the true condition of cylindrical and clubbing bronchiectasis of both lungs. Without the oil no definite conclusion could be had concerning the pathologic condition of the lungs.

This is a composite picture of the right and left lungs taken at two different times.

Diagnosis — Bilateral bronchiectasis

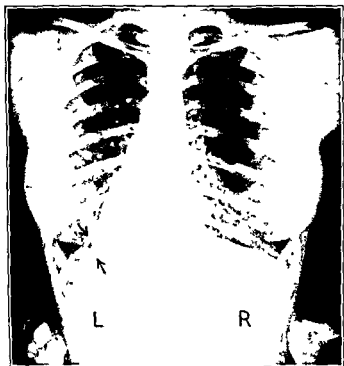


Fig. 7. P.

Diagnosis: Bronchiectasis

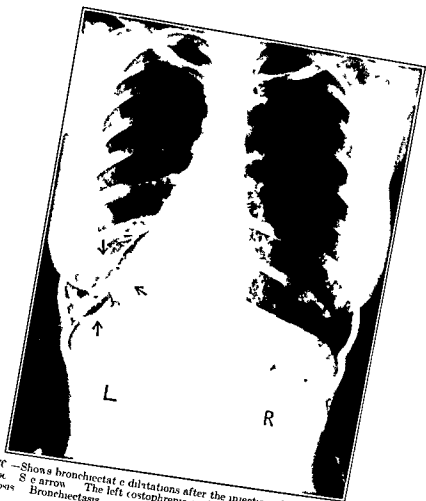


FIG 7C —Shows bronchiectatic dilatations after the injection of iodized oil in the left
 lower lobe. See arrow. The left costophrenic angle is obliterated.
 Diagnosis: Bronchiectasis.

Laboratory Tests—Sputum is an invaluable laboratory material in studying bronchiectasis. It may vary from a negligible amount to over 1500 cc every twenty-four hours. Unless there is marked degeneration of lung tissue elastic fibers are rare. A microscopic examination will show

found since they are the bacteria of the various diseases associated with and predisposing to bronchiectasis. Yeast cells may often be observed. Lymphocytes will not predominate.

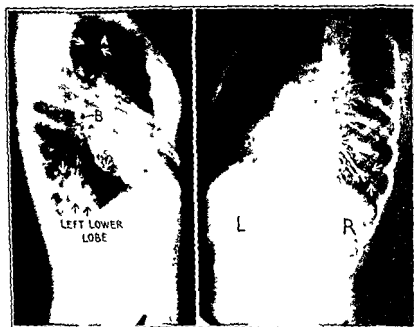


FIG. 77.—A. The left lateral view shows localized sacular dilatations of the left posterior main bronchus. B. Shows a tangential view showing an enlarged bronchus in the right lower lobe. See arrows.

Design 14. Bronchiectasis.

Numerous studies of the sputum for acid fast bacilli should be made both by the concentration method and guinea pig inoculation and culture. Tuberculosis can be mistaken for bronchiectasis very easily, as well as being a complicating disease.

A rapid sedimentation rate test indicates acute exacerbations; it is helpful as an indicator of the virulence of the bacteria present.

Bronchoscopic Examination No case of bronchiectasis or suspected bronchiectasis should be considered fully explored without bronchoscopy. In an advanced case of the disease where secretion is heavy, a bronchoscopy

should precede the roentgen ray examination in order to aspirate purulent material that would prevent the penetration of the iodized oil. The lung in such a case will present to the bronchoscopist evidence of marked lung destruction and cavitation in addition to clearly apparent dilations of the bronchi. Not infrequently a bronchoscopic examination will also reveal a condition causal or complicating condition such as abscess, tumor or foreign body.

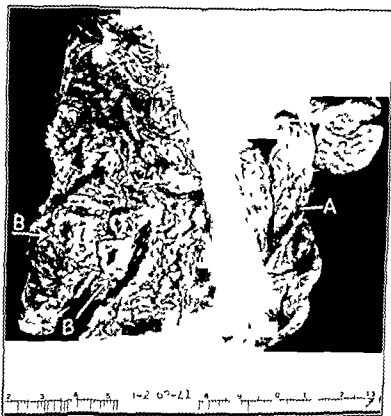


FIG. 78.—Shows the postoperative specimen of the left lung and lingula with widely dilated bronchi throughout. A Shows at let lower. B Shows thickened and dilated bronchi.

Diagnosis: Bronchiectasis left lower lobe.

Bronchoscopy is particularly useful in diagnosing early bronchiectasis where roentgen ray appearances have not become conclusive. In such cases a thickened and redilened area is widespread in the tracheo-bronchial tree mucopus is present. Around the bronchial openings that become at a later stage bronchiectatic this condition will be intensified. It may be noted that these diseased openings do not permit the free passage of air. Holinger
 at each early cases which they call
 important
 bron

Differential Diagnosis—The important diseases to be differentiated

entiation, however, any one of these diseases may occur in conjunction with bronchiectasis or may be complicated by that condition.

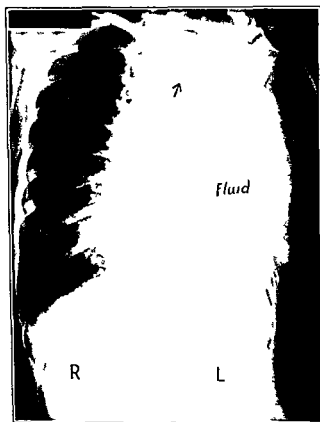


FIG. 79. Same patient as Figure 119. Roentgen ray appearance of the chest after lower left lobectomy. Arrow shows transition to the left which indicates some atelectasis of the remaining upper lobe.

Diagnosis: Post lobectomy for bronchiectatic atelectatic left lower lobe.

Tuberculosis. The existence of dry bronchiectasis previously described places the emphasis in the differentiation of tuberculosis from bronchiectasis at the level of the

should precede the material that would in such a case will destruction and ex the bronchi. Not infrequently a bronchoscopic examination will also reveal a condition causal or complicating condition, such as abscess, tumor or foreign body.

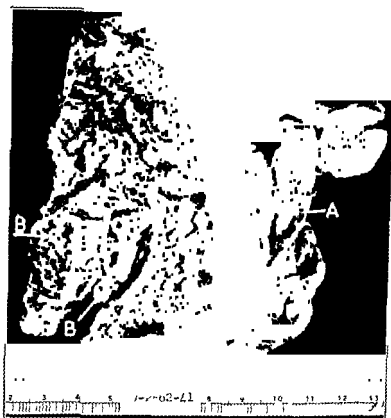


FIG 78 —Shows the postoperative specimen of the lower left lobe and lingula with widely dilated bronchi throughout. A Shows atelectasis. B, Shows thickened and dilated bronchi.

Diagnosis: Bronchiectasis, left lower lobe.

Bronchoscopy is particularly useful in diagnosing early bronchiectasis, where roentgen ray appearances have not become conclusive. In such cases a thickened and reddened area is widespread in the tracheo-bronchial tree, muco-pus is present. Around the bronchial openings that become at a later stage bronchiectatic this condition will be intensified. It may be noted that these diseased openings do not permit the free passage of air. Hohner and De Bakey report cures effected in such early cases, which they call pre-bronchiectatic. This possibility makes bronchoscopy vitally important in all instances where the lingering of symptoms after pneumonia or bronchial obstruction suggests the development of bronchiectasis.

Differential Diagnosis—The important diseases to be differentiated from bronchiectasis are tuberculosis malignancy purulent bronchitis abscess and gangrene of the lung localized empyema fungus diseases and silicosis. The examiner needs to proceed very cautiously in such a differentiation however any one of these diseases may occur in conjunction with bronchiectasis or may be complicated by that condition.

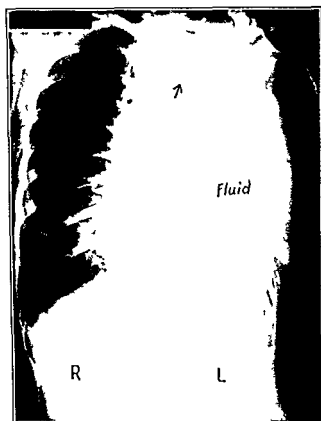


Fig. 73.—Same patient as Figure 119. Roentgen ray appearance of the chest after lower left lobectomy. Arrow shows traction to the left which indicates some atelectasis of the remaining upper lobe.

Diagnosis.—Left lobectomy for bronchiectatic atelectatic left lower lobe.

Tuberculosis. The existence of dry bronchiectasis previously described places the emphasis in the differentiation of tuberculosis from bronchiectasis almost exclusively on sputum tests and roentgenography. In doubtful cases the sputum should be tested for acid fast bacilli repeatedly. If positive evidence is not found in sputum obtained in the usual way, specimens should be aspirated directly from the bronchial tree through a bronchocope.

A roentgen ray study will usually supply important evidence where either disease is present. The area in which indications of disease are observed is significant to diagnosis. Tuberculosis usually occurs in the upper lobes and the lesions are frequently complicated with cavitation and fibrosis. Bronchiectasis is usually located in the lung bases although it may occur in the apices. In lesions of the apex only a conclusive demonstration of the presence or absence of acid fast bacilli can clinch the diagnosis.



Tuberculosis will also present a history of more continuous fever, chills and sweats than bronchiectasis in which these symptoms occur only intermittently and then during pneumonitis attacks. There may also be in tuberculosis a history of contact with infected persons.

Malignancy of the Lung — In this condition there will usually be a history of pain and loss of weight and the duration of symptoms will be much shorter than in bronchiectasis. Hemorrhage and cough are common symptoms to both conditions.

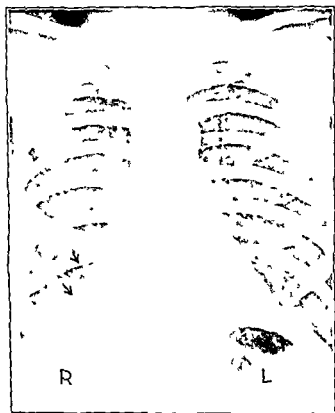


FIG. 81. Malignancy of the Lung. The large, dark, irregular mass in the lower lobe of the right lung is indicative of a malignant tumor. The smaller, dark, irregular mass in the lower lobe of the left lung is also indicative of a malignant tumor.

Roentgenograms will show densities more homogeneous in malignancy

Bronchoscopy is a very important means of differentiating these two diseases. Through the bronchoscope the examiner can actually see any mass or erosions present and he can see any secondary process which might otherwise confuse or obscure the primary situation such as bronchial wall distortion. With a bronchoscope a specimen can be taken of any suspicious mass seen and a biopsy will usually give a positive identification.

However the study of bronchial secretions, especially the secretions aspirated by bronchoscope seems to offer the most effective means of distinguishing the two diseases. Herbut and Clerf in several studies have

p 18 a fuller discussion of the method will be found under Tumors of the Lung p 310. Here it only needs be said that whenever an examiner feels the possibility that either disease is present a bronchial secretions study should be undertaken.

Purulent Bronchitis — This is a relatively rare condition. It may be

Abscess and Gangrene of the Lung — Like bronchiectasis these conditions arise as secondary complications. The usual causes of abscess and gangrene are pneumonia, pulmonary emboli or the aspiration of a foreign substance; these are also important causes of bronchiectasis. However unlike the latter disease abscess and gangrene are almost always an acute condition made by the examiner on the addition to the development of will be higher and sepsis more

acute

A fluoroscopic examination will usually permit a fairly conclusive diagnosis. The abscess condition will show a localized density and possible fibrosis and fluid levels. Lesions will ordinarily be in the middle lobe rather than at the base or apex. The use of iodized oil in roentgenographic studies is complicated however by the fact that the bronchi leading to the abscess are frequently tiny and distorted and thus do not permit the oil to enter into the abscessed cavity. Only occasionally a wide

and

gangrene of the lung occurs
in addition

the base or apex of the lung. A bronchoscopic examination will possibly indicate the condition. roentgenograms with iodized oil following aspiration will usually show the increased density of the abscess above and around the dilated bronchi of bronchiectasis. (See Figure 60 page 117.)

Localized Empyema—These occur frequently as a sequela to the pneumonia. Such a collection of pus is of course so widely different in symptoms and roentgenology from bronchiectasis that confusion cannot usually occur. However an empyema occasionally ruptures into the bronchus and

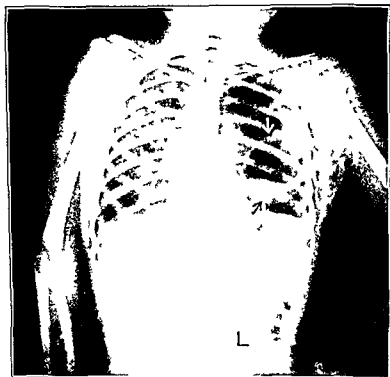


FIG. 87.—N. A. four year 11 white female. The patient entered hospital with a history of a "cold" and x-ray showing growth in the lungs. One year prior to entry. She was born and raised near Reno, Nevada.
Tuberculin 1:1000 negative.

plane angle

Diagnosis: Eccentric cyst of the lung

then considerable difficulty may be encountered in differential diagnosis. Postural drainage and iodized oil injections preceding a roentgen-ray examination will usually eliminate bronchiectasis as a cause, since the bronchi will not be dilated. The injection of air after the aspiration of pus from a localized

empyema will produce a fluid level observable by a fluoroscope study or a roentgen ray film. The injected air will permit a delineation of a local empyema pocket if the patient is made to assume various positions under such examination. (See Figure 3, page 86.)



no air was not injected. The patient was not

long

lost

Fungus Disease This type of infection can only be diagnosed by the discovery of fungi in the sputum or discharging sinuses. Occasionally a

Silicosis—This condition can be readily differentiated from bronchiectasis by roentgenograms except where the latter disease occurs as a complication to the former as it not infrequently does. A roentgenogram of silicosis shows minute densities with increased linear markings beginning at the hilus and extending downward and outward in both lungs. Such markings are usually much more widespread than the enlarged bronchi of bronchiectasis. Dyspnea which starts gradually and increases progressively indicates silicosis especially when there is a history of long exposure to silicious dusts. A more complete description of this disease will be found under Silicosis, p. 215.

BIBLIOGRAPHY

- ADAMS R and CURCHILL, F D Status inversus anust + bronch ecta report of
cases including frequency statistics J Thoracic Surg 7 206 1937
ADAMS W F and ESCUDARO I Aetiology of bronch ecta + its experimental prod-
uction and clinical study Tuberc 19 31 1938
ALFORD A H Tuberculosis of the lower and upper lobes bronch ecta + J Thoracic
Surg 12 28 1943
BAYLIS H C SINGER, J J and GRAHAM F A Bronch ecta + J Thoracic Surg
1 14 1931
BAYLIS C J Ruit
ARD L Henopy + a Hx ch
A M A 82 11 3 1971
of l lct my for l rnel cta +
A L LEMAN J and SKINNER F F Non is gases and l rnel cta
D + of Cl + 13 506 1947
CURCHILL I D Bronch ecta physical and psychological manifestations Nw
Englnd J Med 218 37 1938
EVANS W A JR and CALINIKY I J Diagnosis of bronch ecta + in young adult
patients by pleurocentesis manifest as observed an x ray pers +
An J Roentgenol 51 537 1944
8 118 1938
Invest gation 19
HAYES C The indication technique and results of surgery for bronch ecta + Tr
West Surg Assoc 1937 p 170
HENSELL, H Indication for therapy of early bronch ecta + with reple + cases J
Thoracic Surg 15 23 1946
HERBERT I A and CLARK I H Bronch ecta + J A M A 130 1006
1940
HOLBY C I Relation of bronch ecta + to infection of primary anastomosis Arch Or l
22 537 1933
HUNTER, I H and DEBKEY M Bronch ecta + parts I early bronch ecta +
D (Chest 5 8 1931)
JAMES M Mechanisms of pulmonary abscess pulmonary gangrene and bron-
ch ecta + with regard to method of treatment J Thoracic Surg 8 42 1933
JONES F M JERK W M and WILLIS H S Bronch ecta + of the lung primary +
cl + An J D + 111 72 206 1946
KIN D S Histologic bronch ecta + and its surgical cure Intemat Clin + 11
ser + 47 1947
KINNEY W M Bronch ecta + in pleurothorax D + of Cl + 13 33 1947
KIRBY R S Pathology of chronic tuberculous inflammation of lung Am Rev
Tuberc 35 663 1938

..	.	~	~	..	.	~
					:	.
					:	.

24. BRONCHOPNEUMONIA

BRONCHOPNEUMONIA is a term used to designate a whole group of infections with a somewhat varied etiology but with pathologic similarities. In a large percentage of such pneumonias it is impossible to identify the particular bacterial agent. Of the balance streptococcal bronchopneumonia is by far the commonest. Staphylococci are responsible for some cases, and the Friedlander bacilli for others. The pneumococci important to lobar pneumonia occasionally are found in bronchopneumonia; the latter condition is much more likely to result from an invasion by the higher numbered types which are ordinarily not pathogenic.

Etiology—The micro-organisms that usually cause bronchopneumonia are very commonly present in the throat of healthy people. The pneumococcal bronchopneumonia is caused by the most common inhabitants of the oral cavity. From this it would seem evident that the major factor in the etiology of this pneumonia is body resistance to infection. This is substantiated by the age incidence of the disease—it is most often found in the very old or very young. It is frequently the terminal event to the process. In the young it frequently occurs after infectious exanthema.

This factor of body resistance may also account for the frequency with which an epidemic of bronchopneumonia of one type or another follows an epidemic of influenza. However, the factor in the instance of pneumonia following other infection is probably more intimate; the pneumonia probably actually represents a secondary infection.

Direct predisposing causes besides other diseases are varied. Bronchopneumonia is frequently followed by chemical injury to the epithelium, such as ether anesthesia and also by irritant gases in warfare or in certain manufacturing plants. Trauma may weaken a local area of tissue and prepare the way for a bacteriologic invasion. Aspiration of foreign objects, such as food, may induce bronchopneumonia, as may submersion (drowning). Most important of all is sudden chill. The reason a chill may be followed by lobar pneumonia in one instance and bronchopneumonia in another is a mystery still unsolved.

In certain instances, particularly following an influenza epidemic, bronchopneumonia may itself become contagious and epidemic.

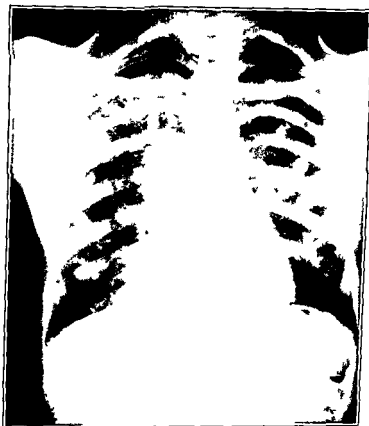
Pathology The varied bacteriology of bronchopneumonia of course implies that there can be no uniform pathology, but there are certain features typical of the whole group of infections. The process seems to start almost always in the bronchi, which become inflamed. An exudate develops, but it is seldom present in great quantity. This exudate, which is purulent,

The exudate
in the alveoli

Upon inspection of autopsy specimens of the lung the most obvious feature is the focalization of the infections with consolidates that may

include in the same section of the lung polymorphonuclear leukocytes fibrin serum or erythrocytes. The lung tissue in between these patches may be healthy or it may be emphysematous or atelectatic.

In most instances the patches of infection have little tendency to coalesce but occasionally a whole lobe or even a lung may become consolidated.



phrenic angles

Diagnosis: Pulmonary edema and bronchopneumonia

A rather frequent finding in bronchopneumonia is the development of small multiple abscesses. This is especially likely to occur if the infecting organism is the streptococcus or Friedländer's bacillus.

Friedländer pneumonia is rather like lobar pneumonia in its pathology. It tends to be confined to one lobe although it is frequently bilateral. Engorgement is the typical appearance. Necrosis is often widespread.

Bronchopneumonia generally is a slowly resolving condition with fibrosis and consolidation in areas often becoming permanent.

Clinical Symptoms—In instances where bronchopneumonia is a sequel to other diseases such as bronchitis, measles or influenza the separation of the symptoms of one from those of the other is impossible. Where it attacks a healthy person the symptomatology is fairly typical.

Onset is usually sudden but less so than in lobar pneumonia. Chills, malaise and fever are usually first noted. Cough is usually much more severe than the cough in lobar pneumonia and is almost always present.

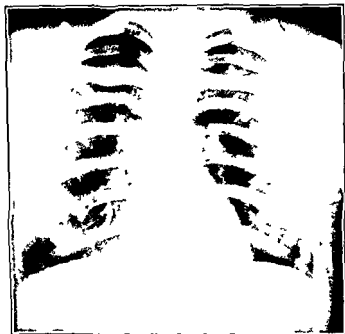


Fig. 83. Same patient as Figure 81. On 3-2-43 the roentgenogram disclosed complete resolution of the consolidation at the normal chest roentgenogram.

Diagnosis.—Clearing of chest following lobar pneumonia.

Dyspnea is also very common. Pain in the chest over the lesions may or may not be present. Cyanosis may develop as the disease becomes full-blown. There will always be sputum but it may be purulent, clear or bloody. It will not be the rusty sputum of lobar pneumonia. Breathing may be rapid and shallow. Fever may be quite extreme at times but it will be irregular. Marked anorexia may be present.

Physical Signs—Bronchopneumonia is oftentimes so scattered that present. Some impairment of chest expansion will be seen.

Roentgen ray Findings The areas of bronchopneumonia will appear on a roentgenogram as patches of increased density. They are very often round. They may frequently be seen to shift location possibly due to healing in one part and infection of another but more likely representing edematous areas. In addition areas of emphysema sometimes resembling air cysts may be seen. Darker shadows of small areas of atelectasis are common. The diaphragm will often be raised. Where massive atelectasis occurs the mediastinum may be radically displaced. Mediastinal lymph

are very important to prognosis.

Bronchoscopy This procedure is seldom advisable in patients with bronchopneumonia because of the possibility of further injury to an already inflamed bronchus.

Laboratory Findings Wherever possible the specific organism responsible for the pneumonia should be identified since they vary in susceptibility to the various sulfa drugs streptomycin and penicillin. The occurrence of one of them in the blood stream is the best indication of the cause of a particular type of bronchopneumonia but this is a rare finding.

The character of the blood itself is much like that of lobar pneumonia. Red blood cell count and hemoglobin are practically normal with a high sedimentation rate, high leukocyte count and a predominance of polymorphonuclear cells. The leukocyte count and sedimentation rate however are usually somewhat less than those of lobar pneumonia.

Differential Diagnosis—Bronchopneumonia will be most difficult to diagnose when it is a sequel or secondary to another disease. It should be considered whenever there is a sudden shift for the worse in a seriously ill patient particularly if the patient is very young or elderly.

A primary bronchopneumonia before roentgen ray examination may be confused with the same conditions that lobar pneumonia is they will be discussed later. However even with roentgenograms a primary bronchopneumonia may be confused with lobar pneumonia, tuberculosis or lung abscess.

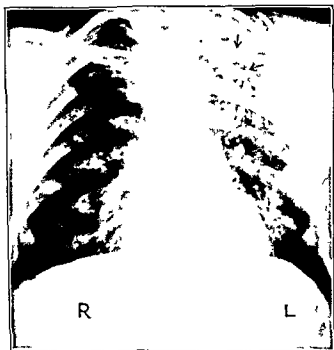
Lobar Pneumonia—Bronchopneumonia caused by the Friedlander bacillus is the only form of that disease which physical signs and roentgen ray appearances will not easily differentiate from lobar pneumonia. This bacillus rather frequently produces a homogenous consolidation in one lung. A close examination will usually reveal the disease as not conforming in its extent to the lobe, it is usually bilateral and the sputum is frequently

stration of the specific organism in the sputum.

Another ready source of confusion is the fact that lobar and bronchopneumonias may both be caused by pneumococci. The bronchopneumonia is ordinarily found in the aged and in infants. Its onset is usually less abrupt than either lobar pneumonia or the other bronchopneumonias.

Fever will be irregular. A roentgen-ray examination will be the basis of differentiation. (See Figure 29, page 71.)

Tuberculosis—Bronchopneumonia is even more apt than lobar pneumonia to be difficult to distinguish from tuberculosis since bronchopneumonia lesions are frequently apical, particularly with the Friedländer



bacilli as agents. The scattered patchiness is also suggestive of tuberculosis. Where the respiratory and circulatory distress in severe bronchopneumonia is likely. Sputum studies for the specific micro-organism should be made. Leukocytosis indicates a process other than tuberculosis is present. The two may coexist.

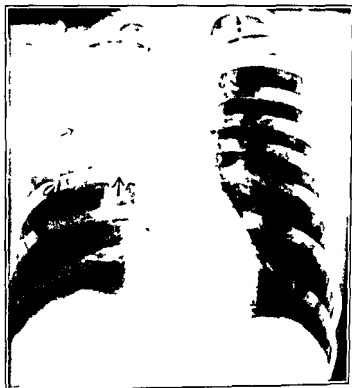
Pulr " " " "
of the
found

reveal scattered patches of inflammation elsewhere in the lungs. The breath of a patient with bronchopneumonia will not be as foul as that of a

patient with lung abscess. Where diagnosis depends upon sputum analysis the test is the demonstration of anaerobes, since the bacteria of the bronchopneumonia will often be present with cavitation.

R

I



L

ed

negative for acid fast bacilli

Diagnosis: Chronic lung abscess with chronic pulmonary tuberculosis (?)

BIBLIOGRAPHY

- AMBERSON J B JR Aspiration bronchopneumonia Internat Clin 3 126 1937
 BULLOW J G M Management of Pneumonias New York Oxford Medical Publications 1937
 BULLOW J G M and GREENBAUM I Pneumococcal pneumonia in infants and in children Am J Dis Child 53 22 1937
 JAY C H C Surgical Diseases of the Chest Philadelphia with streptococcal Allergic Bronchopneumonia Am J Dis

25. LOBAR PNEUMONIA

Although there are actually many kinds of bacteria that produce pneumonia, the pneumococci account for about 85 per cent of the cases encountered. Thirty-three types of pneumococci have been identified and there remains a small unclassified group but only a few of these types are common. The remaining 15 per cent of the cases of pneumonia are caused by *staphylococcus aureus* or *staphylococcus albus*, *bacillus Friedlander* and some viruses. These agents produce pneumonias pathologically quite different from lobar pneumonia and will be discussed separately in the following chapters.

The use of penicillin and sulfa drugs in recent years has not only greatly decreased the mortality of pneumonia but has actually decreased its incidence particularly by the diminution of post-operative pneumonias. Lobar pneumonia however is still a common disease and it is still not infrequently fatal so early diagnosis is important.

Etiology The fact that some of the pneumococci are almost always present in the throat of healthy individuals indicates that resistance to lung infection is normally present. The epiglottitis is probably the most important mechanism in this resistance but the ciliary action of the trachea and larger bronchi is also effective in keeping the lungs clear.

A sudden chill is the most important factor in lowered resistance to pneumonia. It is shown not only by the frequent number of cases occurring during the winter months. Robertson has shown that a sudden chill in a dog results in incomplete closure of the glottis. It is likely that this is an important factor in the etiology of lobar pneumonia.

That the ciliary action of the bronchi is also an important mechanism in the etiology is pointed out by O'Hara. Anything that irritates the bronchi interferes with their function definitely predisposes to pneumonia. Tobacco smoking is in example. The high incidence of post-operative pneumonia that formerly obtained was probably due in large measure to the irritation of the membrane by the anesthetic. The tendency of the common cold to predispose to lobar pneumonia is probably due in large measure to the same factor.

Malnutrition, fatigue, alcoholism and the presence of other diseases may also lead to lobar pneumonia by lowering physiological resistance to infection generally. Also the occurrence of pneumonia so much oftener in males than females at all ages suggests some factor inherent in the constitution of the individual. This factor of active resistance is a matter of fact probably the key factor with the non-mechanical factors of predisposition leading to lobar pneumonia by weakening the inherent resistance.

An unexplained etiological feature of the disease is the variation that exists in susceptibility to different types of pneumococci among the different age groups and between the sexes. Type I is the most frequent agent of lobar pneumonia in all groups. Type II however accounts for a much

Contrary to earlier thought pneumonia is a contagious disease. Because of the resistance factors we have discussed are ordinarily very efficient in instances in which it has been possible to demonstrate the source of contamination are uncommon. However it is often possible to demonstrate the infecting bacteria in the throats of those who are in close contact with a patient with lobar pneumonia. The existence of carriers of pneumonia has been established.

Pathology — Most authorities agree that the pneumococci are probably carried into the lung in a droplet of saliva and are therefore inhabitants of the throat of the patient for at least a short time preceding infection. Attempts to produce pneumonia experimentally with air borne pneumococci have been unsuccessful. For them to become pathologically active and multiply they must be within some other material such as the secretions of the throat. They may be carried through the smaller bronchioles and into the alveolar sacs by gravity. On the other hand it is possible that they penetrate the larger bronchi and spread through the interstitial tissue. The lymphatics are usually invaded very early and are thought by some authorities to be important in the spread of lobar pneumonia.

Earlier thought based on necropsy examination was that the pneumonia began at the hilus and advanced outward into the lung. Serial roentgenologic studies have shown that the process begins at the periphery of the lung and progresses centrally. It is a fact however that the lungs of pneumonic patients at autopsy almost invariably show the extensive involvement to be hilar.

The first reaction of the lung to the disease is engorgement. The interlobular veins themselves are followed by a sputum char with the abundance red liver like on the tissue reaction many free

leukocytes and much fibrin.

As the engorgement recedes and the deposited leukocytes and fibrin are broken down the lung becomes gray and almost crumbly.

That little real damage is done to the deeper tissues is demonstrated by the rapidity with which the lung regains its normal appearance and function after the infection has been arrested. The leukocytes and fibrin degenerate and are absorbed by the circulating blood. In less than 10 per cent of the people who contract lobar pneumonia is there permanent lung damage in the form of fibrosis.

In the majority of patients lobar pneumonia is confined to one or two lobes usually unilaterally. The lower lobes are by far the most common site of infection. A frequent finding is an infection that includes the whole

lower lobe and a thin slice of the lobe above. The demarcation between healthy and invaded tissue is usually quite sharp. It has been experimentally demonstrated that this distribution is in accordance with bronchial anatomy. It is entirely possible that lobar pneumonia spreads largely by infected edematous fluid through the bronchial tree as this distribution would indicate.

Occasionally the mucus from the infected lobe may become lodged in

complications have been reduced enormously by their use. Septicemia was

sulfis and biotics

will be preceded by
ms of the pneumonia
om is a severe chill

which is followed shortly by a rapidly developing fever, dyspnea and localized pain. The pain is usually severe. It is due to the involvement of the pleura. It is most often experienced directly over the lobe involved but may sometimes confuse diagnosis by being referred to the abdomen or the shoulder of the patient.

Fever is more constant in pneumonia than in most other infections. After the initial sharp increase in temperature it usually rises very slowly for from five to seven days until the crisis is reached and then falls abruptly.

Respiratory and circulatory disturbance is usually severe. Breathing is rapid but shallow where pleural pain is intense it may further aggravate respiratory difficulty. Because of the serious reduction in air intake due to this symptom in addition to the portion of the lung edematous and non-functioning cyanosis is a prominent and early-developing symptom. It is most pronounced in the earlier stages of the disease.

The nervous system is often affected rather seriously with symptoms ranging from severe headache to delirium and violence. Occasionally one finds a patient in a state of shock that is much like severe surgical shock and equally fatal. The nausea and vomiting that frequently accompany lobar pneumonia may be due to the reaction of the nervous system or it may be toxic in origin.

Cough is usually present at some time during the course of the infection. The character of the cough as dry, harassing, paroxysmal, etc. will be dependent upon the viscosity and amount of the sputum that escapes into the bronchi. Sputum of almost every description may be present at some time or another but a rusty sputum is characteristic of the stage of red hepatization which is also the stage in which the disease is usually diagnosed. Hence rusty sputum is of great diagnostic importance. A frankly hemorrhagic sputum is rare. Amounts are usually rather small in comparison to other lung infections.

Physical Signs — Diminished resonance over the affected side is an early sign in lobar pneumonia. The note becomes duller as the consolidation becomes more complete. Bronchial breathing is a characteristic also of such consolidation.

absorbent tissue

breath sounds

pitched rales are a usual finding

The areas of atelectasis will be marked by a flat note elicited upon percussion much like that elicited over fluid. Where the pleura is involved as it usually is, a scratching friction rub may be audible. If a free pleural exudate develops in the course of the disease it will obscure the physical findings in the lung itself.

Roentgen ray Findings — Abnormalities can be seen on a roentgenogram of the chest from six to twenty-four hours after the onset of symptoms in a patient with lobar pneumonia. As already noted, congestion first appears at the periphery of the lung and works in. However, this congestion frequently begins at the central anterior or posterior of the lungs and may from anterior-posterior view appear to be a triangle with its base at the hilus. A lateral view, however, will show the consolidation is being more extensive peripherally.

The shadow in the disease becomes denser and more extensive as the disease progresses. It will not, as sometimes thought, always involve the entire lobe, however. As before remarked, the area involved seems to coincide with the area served by a particular bronchus or bronchial system. The diseased tissue will ordinarily be rather sharply demarcated from healthy tissue, and the line of demarcation will usually be straight rather than curved. Lobar pneumonia will not achieve its maximum density before the third day after the onset of symptoms.

In early stages of lobar pneumonia the shadow may lie completely behind the heart shadow on an anterior-posterior roentgenogram. Such a condition can be detected by the increased density of the shadow on one side of the mediastinum.

Bullow points out that various types of pneumococci may produce different appearances. Such differences may be significant to prognosis. The

some such displacement exists it will be toward the affected side. The diaphragm on the affected side will normally be raised, and a fluoroscopic examination may show it to be much less mobile than the normal side. (See Fig. 29, page 71.)

most numerous in the clumps of rusty sputum. Agglutination should be

spring should remain
ce penicillin and the
seldom do it. How

ever, in hospitals where laboratory facilities are at hand, typing offers information of value to the physician in determining prognosis and treatment.

The blood should always be checked for evidence of bacteriemia. It will otherwise present a rather typical picture of a septic process. The red blood cell count will be normal or nearly so, but the leukocyte count will be greatly increased in most cases. Sixteen thousand to twenty thousand is a usual finding, with counts as high as sixty thousand occasionally encountered, especially in children. A low leukocyte count indicates a bad prognosis. The

ever-present tuberculosis must be ruled out first of all in a diagnosis of lobar pneumonia. Bronchopneumonia must be considered. Pulmonary infarct sometimes may be mistaken for pneumonia as may atelectasis. The whole group of pleural disorders—acute fibrinous pleurisy, serofibrinous pleurisy, and empyema—may be mistaken for lobar pneumonia to which they are often secondary. Occasionally the respiratory and circulatory distress of the patient will suggest congestive heart failure or coronary occlusion. In a few instances the referred abdominal pain has been so severe as to lead to a diagnosis of appendicitis or some other acute abdominal condition. Occasionally massive carcinoma of the lobe may simulate lobar pneumonia.

Tuberculosis. As Bullowa points out, patients with a history of several months' illness with loss of weight and frequent colds are not immune to pneumonia. When such patients are encountered the onset of pneumonia may easily be mistaken for an acute form of tuberculosis. This is particularly likely to happen when the pneumonia is in an upper lobe. However, the sputum in tuberculosis is likely to be frankly hemorrhagic, which is practically never the case in pneumonia. The fever in tuberculosis is much more irregular and tuberculosis will almost always involve a greater lung area than pneumonia. Whenever positive differentiation is not possible on the basis of roentgenologic, physical and clinical evidence, a persistent effort to find both kinds of bacteria in the sputum should be made, because the two conditions not infrequently are found in the same patient. Leukocytosis will not be present in tuberculosis alone.

Bronchopneumonia.—Only rarely does bronchopneumonia infiltrate an entire lobe, and when it does patchy shadows in other areas are usually visible roentgenologically. The demonstration of the micro-organism responsible will usually make diagnosis possible. Bronchopneumonia is almost never so symptomatically abrupt as lobar pneumonia is. (See Fig. 84, page 170.)

Pulmonary Infarct. Pulmonary infarct is also a condition whose onset is sudden. Confusion may arise because it is a post-operative complication as lobar pneumonia may be, and the roentgenologic evidence may not be

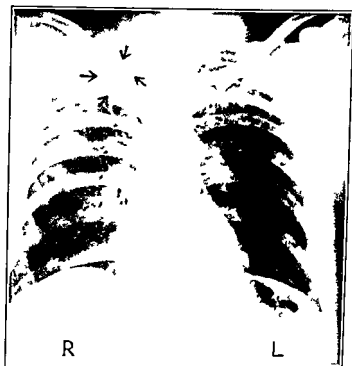
shock rather than sepsis.

Atelectasis D

nose throat or a

severe chest pi

hemorrhagic The roentgen ray examination will reveal a shadow that is more homogeneous than that of pneumonia The homolateral diaphragm will be much higher than is the case with lobar pneumonia and mediastinal



and tracheal displacement will be prominent Atelectasis is also encountered with similar symptoms following non surgical obstruction of a bronchus but history will usually suggest the cause of the symptoms

Acute Fibrinous and Serofibrinous Pleuritis —When an acute pleurisy arises abruptly it may be difficult to know whether or not it is secondary to lobar pneumonia The physical examination will show difficulty in breathing but none of the physical signs of consolidation such as bronchial breathing The roentgen ray examination will show the lungs clear

Serofibrinous pleurisy will ordinarily not be confused with lobar pneumonia because its onset is usually less abrupt, and its symptoms are milder. The percussion note will be flat rather than dull over free fluid. Breath sounds will be absent, or greatly diminished. Roentgenologically there may be shadows somewhat like the shadow of lobar pneumonia but the

R

L



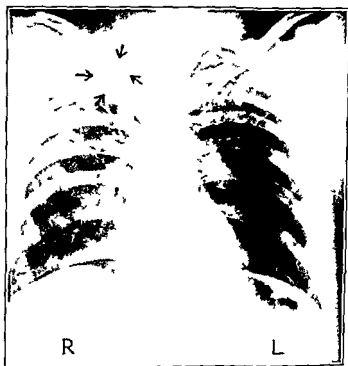
mild arrow

Diagnosis: Left apical cavity and consolidation of left lobe and partially left diaphragm
Tuberculosis

fact that such a shadow is not strictly confined to a lobe will be readily apparent. Diagnostic puncture should be made. (See Fig. 24, page 65.)

Empyema—This condition is more apt to present the rapid onset with a high degree of sepsis which is characteristic of lobar pneumonia. Roentgenologic distinction may be difficult where the empyema is unilateral

Atelectasis — Post-operative atelectasis follows surgical operations on the nose, throat or abdomen. It is heralded by an acute rise in temperature and severe chest pain as is pneumonia. Expectoration however is usually hemorrhagic. The roentgen ray examination will reveal a shadow that is more homogeneous than that of pneumonia. The homolateral diaphragm will be much higher than in the case with lobar pneumonia and mediastinal



and tracheal displacement will be prominent. Atelectasis is also encountered with similar symptoms following non surgical obstruction of a bronchus but history will usually suggest the cause of the symptoms.

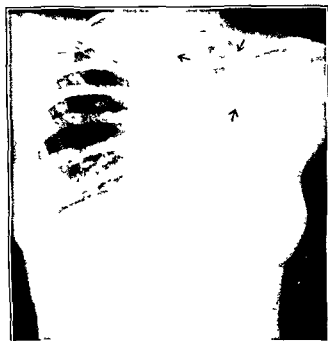
Acute Fibrinous and Serofibrinous Pleuritis — When an acute pleurisy arises abruptly it may be difficult to know whether or not it is secondary to

Serofibrinous pleurisy will ordinarily not be confused with lobar pneumonia,

may be shadows somewhat like the shadow of lobar pneumonia, but the

R

L



mid arrow

Diagnosis Left apical cavity, atelectasis lower left lobe and paralyzed left diaphragm

Tuberculosis

fact that such a shadow is not strictly confined to a lobe will be readily apparent. Diagnostic puncture should be made. (See Fig 24, page 65.)

since empyema is often much less widespread than serofibrinous pleurisy. Pneumonia will be indicated, however, by the physical signs of bronchial breathing, increased whisper and voice sounds, and dull rather than flaccid sounds on percussion. Diagnostic puncture of the pleural cavity will aid in diagnosis.

Congestive Heart Failure and Coronary Occlusion—Both of these conditions may present fever and signs of sepsis much like those of a moderate

R

L



essentially normal.

The patient expired of massive hemorrhage. Post mortem examination revealed squamous cell carcinoma.

Diagnosis: Carcinoma of the lung.

pneumonia and occasionally there will be moderate physical findings over a lower lobe. The roentgenologic findings may show some lung disturbance but the shadows will not be so clear-cut as those of pneumonia. Temperature will be irregular. Failure to demonstrate pneumococci in significant amounts, and severe respiratory and circulatory distress with only moderate lung involvement may suggest coronary disturbance. An electrocardiogram will confirm or refute such a suspicion.

Appendicitis—Pain from the pleura in lobar pneumonia is rather

frequently referred to the abdomen. It may be severe enough to produce The occasional other abdominal examination

including roentgenograms of all patients with acute abdominal symptoms

Cancer of the Lung — A single massive tumor may occasionally be encountered that roentgenologically resembles lobar pneumonia. Such a tumor because of the insidious non acute nature of its symptoms is not likely to be mistaken for lobar pneumonia unless it comes to the attention of the physician because of some other acute inflammation. It should be considered if a pneumonia behaves peculiarly and especially if hemorrhagic rather than rusty sputum is coughed up. Diagnosis can be made on the basis of stain studies of such sputum or by bronchoscopic study including biopsy specimens.

BIBLIOGRAPHY

- BARACH A I. Acute disturbance of lung function in pneumonia in the light of oxygen treatment. *J A M A* 89:1863, 1927.
- BULLOWA J G M. Management of pneumonias. New York: Oxford Medical Publications, 1937.
- massive atelectasis of the lung. *Bull New York Academy of Med.* Second series 4:384, 1928.
- Size of the consolidated lung in lobar pneumonia. *Am J M S.* 178:15, 1922.
- FRIED B M. Allergic inflammation of lungs: pathogenesis of lobar pneumonia. *Arch Path* 18:86, 1934.
- FRISCH A W. Sputum studies in pneumonia: in vivo and in vitro susceptibility of pneumococci to sulfapyridine and sulfathiazole. *Am J Clin Path* 11:737, 1941.
- FRIED A W and RICE A F. Sputum studies in pneumonia: selection of therapy. *Ann Int Med* 15:387, 1941.
- GRAHAM F A, SINGER J J and BALLON H C. Surgical Diseases of the Chest. Philadelphia: Lea & Febiger, 1935, p. 704.

PULLEN R L. Tuberculous pneumonia: review of American literature and report of

26. LIPOID PNEUMONIA

SINCE the first report of this condition by Hughlin in 1923 lipoid pneumonia or oil aspiration pneumonia has attracted much attention because of its unusual manifestations. It has been reported with frequency in the larger clinics and as its characteristics become more widely known it may prove to be a more common condition than its fairly recent discovery would indicate.

Etiology—In the majority of instances such oil is taken orally and hence the pneumonia occurs most frequently in those groups where the swallowing mechanism is apt to be defective—infants, the aged and the bed-ridden. Oil aspiration is also reported in patients with neurological lesions involving the swallowing reflex.

The second route for oil aspiration is nasal and results from the protracted use of oily nasal medications. Lipoid pneumonia may occur in patients habitually using oil sprays where there is no involvement of the glottis.

In children lipoid pneumonia may occur from the aspiration of a wide variety of oils—animal, vegetable and mineral. The most extensive pathology results from the aspiration of mineral oils. In adults petrolatum is almost always the irritating agent and the vegetable oils except for chaumogra oil do not ordinarily produce much reaction. Only one or two instances have been reported in which oils used for roentgenoscopy such as poppyseed oil have been causative factors.

A so-called acute lipoid pneumonia has been identified. In this condition the primary pathologic agent is bacteria. It results from the use of nasal oil sprays for sinus and tracheal infections. The oil droplets irritate the parenchymal tissue to an edematous state especially favorable to infection and in addition probably carry the bacteria to the lung. The condition is not actually lipoid pneumonia.

True lipoid pneumonia is ordinarily the result of the habitual use of irritating oils over a period of years. Jones however reports two cases in which lipoid pneumonia apparently followed the use of nasal oils for a very short time.

Pathology—The initial response of the lung to the presence of oil in the alveoli is an exudate of mononuclear and polymorphonuclear cells. This reaction is usually mild. Phagocytes appear in large numbers and ingest the oil droplets. These so-called lipophages are the most characteristic microscopic feature of this pneumonia. The droplet of oil is usually distinctly visible within the cytoplasm of the cell. The gross appearance at this stage is of a foamy white oily liquid within the alveoli.

lipophages. Pulmonary arteries usually show intimal atheromatous changes and medial hypertrophy in the small branches. The alveoli may become lined with a continuous cuboidal epithelium.

Fibroblasts appear and gradually the inflamed areas are replaced by a dense collagenous connective tissue. This tissue closely resembles tumor tissue except for the small pockets of entrapped oil that are visible on cut section.



FIG. 91.—Lipoid pneumonia specimen. Right lung sagittal section showing fibrosis of the lower lobe and portions of the upper and middle lobes.

A. Shows large dilated bronchus.

B. Pneumonic consolidation.

C. Dilated bronchus with unusually thickened wall.

Diagram. Lipoid pneumonia and bronchiectasis. Post mortem specimen.

Since lipoid pneumonia is caused by the aspiration of small amounts of oil over a long period of time, the lung may show all the stages of pathologic development simultaneously. The most marked changes are usually in the lower lobes because of gravity. The characteristic posture of the patient after aspiration of the oil is the determining factor. In many instances the lesions are widespread, with lipophages also found in areas of compensatory emphysema.

Lipoid pneumonia is rarely directly responsible for the death of a patient but because it severely modifies lung function it frequently predicates to purulent bronchitis and recurrent attacks of bronchopneumonia and bronchiectasis.

Clinical Symptoms The symptoms of lipoid pneumonia are apt to come to the attention of a physician because of their persistence rather than because of their acuteness. The most common are moderately productive cough, intermittent fever and rather slight pain in the chest. Lipoid pneumonia of subclinical symptoms or asymptomatic is occasionally suggested by routine roentgen examination. Severe symptoms of high fever and chills indicate a secondary infection. Blood tinged sputum is not unusual.

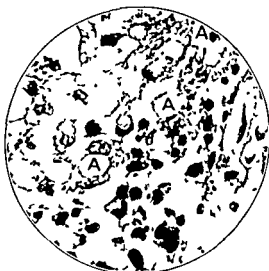


FIG. 92.—Lipoid pneumonia. Microscopic section. Mononuclear cells containing fat globules marked with A.

There is progressive weakness and loss of weight. There is some tendency for the symptoms to be cyclic. Where the patient has dysphagia, severe strangling attacks of coughing during or after meals suggests that food is being aspirated into the trachea.

Physical Signs There are no pathognomonic physical signs of this disease but in many instances dullness upon percussion and variations in breath sounds, with or without rales, can be detected. The presence or absence of such signs is dependent upon the amount of involvement of the lungs and the location of the disease process.

Roentgen ray Findings—There are no roentgenologic shadows which normally coalesce into large shadows which may include a whole lobe. Compensatory emphysema may be noted at times in the uninvolved portions of the lung.

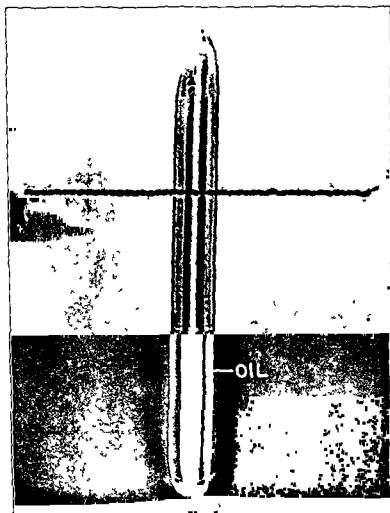


Fig. 93.—Oil extracted from the lungs of a patient with lipoid pneumonia at autopsy (approximately 0.5 cc.)

These signs of lipoid pneumonia are most commonly seen in the bases of the lung and only rarely in the apices. The right lung is somewhat more apt to be involved than the left. Except for the gradual atelectasis of heavily infiltrated areas the roentgenograph of an infected lung may show no changes over a period of years if the patient no longer aspirates oil.



FIG. 94.—The left lower lobe is atelectatic with compensatory emphysema in the costophrenic space. There is a pronounced triangular shadow in the right lower lobe with striated shadows below and a high diaphragm. See arrows.
Diagnosis: Lipoid pneumonia and bronchiectasis.

Laboratory Tests—Diagnosis of lipoid pneumonia before autopsy is most frequently made by the demonstration of oil droplets consistently in the sputum. Conclusive diagnosis will depend upon laboratory determination of the oil in the sputum as exogenous. Since oil is fairly persistent in the pharynx the patient must not take oil orally for several days preceding the collection of sputum specimens.

When uncomplicated by secondary infections lipid pneumonia does not produce marked changes in the blood

Bronchoscopy—Bronchoscopic visualization of the affected passages is seldom very helpful. Diffuse inflammation will be seen and some constriction of the involved bronchi may be noted. Hilar fibrosis may be present but its nature is non specific. If however bronchoscopic aspiration of fluid from the inflamed bronchi is done oil droplets are often discovered in such fluid

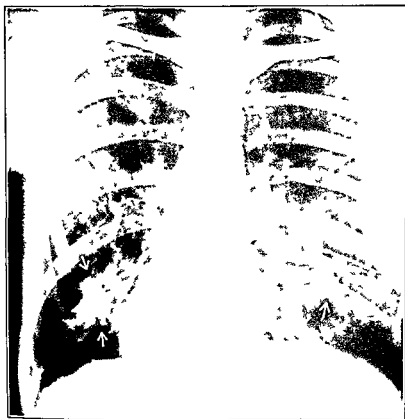


FIG 93.—Film made before the injection of iodized oil. Injection of the oil clearly indicated the diagnosis. (See Figure 74, page 156)

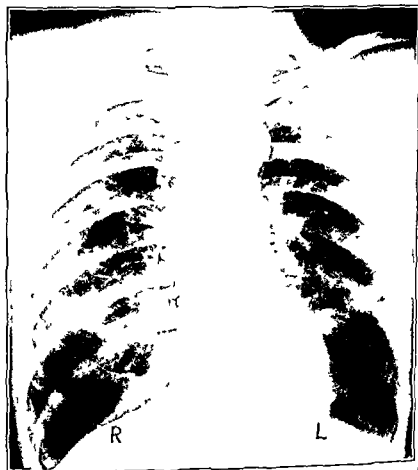
Diagnosis: Bilateral bronchiectasis.

Differential Diagnosis—Lipoid pneumonia may be mistaken for almost any lung disease that is characterized by irregular consolidation. It is most commonly mistaken for silicosis, carcinoma, bronchiectasis with recurrent pneumonitis, and fungus disease.

Silicosis—This disease is also characterized by linear and nodular fibrosis. However, in silicosis the fibrosis is ordinarily much more widespread.

with every part of both lungs affected. In practically every instance a history of prolonged exposure to pathologic dust can be elicited. (See Fig. 112, page 221.)

Carcinoma—It is oftentimes impossible to differentiate carcinoma and lipoid pneumonia on the basis of physical signs and roentgenograms. Local



ized pain, frequent hemoptysis and rapid physical deterioration suggest carcinoma. Where lipoid pneumonia cannot be absolutely confirmed on the basis of oil in the sputum and a history of oil aspiration biopsy may be necessary to exclude carcinoma. (See Fig. 34, page 81)

likely to
Sputum
injection
t to occur
ion which

the author has encountered several times. (See Fig. 45, page 189)

Fungus Disease—Only the higher bacteria-like fungi such as the *Aspergillus* genus produce the linear fibrosis typical of lipoid pneumonia. These fungus infections of the lung are most apt to be accompanied by cutaneous lesions or bone erosion. Differentiation when cutaneous or osseous lesions are not present must depend upon the recognition of fungi or oil droplets in bronchial secretions.

Pulmonary Tuberculosis—Lipoid pneumonia has not infrequently been diagnosed as tuberculosis and the possibility must be considered in diagnosis. The demonstration of acid fast bacilli or of oil droplets in the sputum is of course necessary for positive differentiation. A history of the use of oily medicaments is almost always to be elicited in adults with lipoid pneumonia and will mitigate against wrong diagnosis. (See Fig. 14, page 273)

BIBLIOGRAPHY

- BISHOP, P. G. C. Oil aspiration pneumonia and pulmonary lipoidosis. *Ann. Int. Med.* 13, 1327, 1940.
- BROWN, A. I., and BISHOP, G. R. Differential diagnosis between lipoid pneumonia and pulmonary neoplasm, report of case—treatment by partial lobectomy. *J. A. M. A.* 117, 4, 1941.
- LEIDA, K. Lipoid pneumonia of adult type (paraffinoma of the lung)—report of five cases. *Arch. Path.* 23, 470, 1937.
- JAMES, R. M. Lipoid pneumonia simulating bronchogenic carcinoma. *J. Thoracic Surg.* 16, 451, 1947.
- FATCHER, G. F. Studies in pneumonia following nasopharyngeal injections of oil. *Am. J. Path.* 1, 407, 1925.

27. VIRUS (ATYPICAL) PNEUMONIA

THE successful treatment of pneumonia has had shown not only in recent years but also by the increasing percentage of virus pneumonia reported by the larger hospitals. This latter phenomenon may indicate an actual increase in incidence on the other hand it may merely indicate that a large number of virus pneumonias were wrongly diagnosed as bacterial pneumonias until chemotherapy underlined the distinction.

The increased attention to the viruses has already been fruitful but our knowledge is far from complete. Much progress has been made in the identification of specific viruses in experiments with animals but the factors of transmission and so on are still speculative. In their attack upon higher organisms the viruses are thought to be intracellular rather than intercellular. This probably explains both their failure to react to chemotherapy and the failure of the patient with a virus pneumonia to show the typical reaction of leukocytosis to a pathogenic invasion.

Etiology—Primary atypical pneumonia is the most common of the virus pneumonias. It is often found to occur in small epidemics hence it seems likely that it can be passed from one person to another. The sporadic nature of such outbreaks is however unexplained and no habitation of the virus outside the human body has been demonstrated. Most viruses are known to increase in virulence as they are passed from one carrier to another and it is possible that the virus responsible for the clinical entity primary atypical pneumonia is a normal inhabitant of the human throat or at least that healthy carriers of the organism exist. The disease occurs primarily in the winter months and a large percentage of persons contracting the disease are young adults. The bodily factors influencing contraction and severity and so forth are of course unknown.

Influenza pneumonia accompanies epidemics of influenza and is caused by the same organism. It was suggested that the responsible agent was a virus during the great outbreaks in 1918-20 but the responsible virus was not isolated and identified until 1935 when a large filterable virus about 100 microns in diameter was found to produce influenza in ferrets. Like other viruses it is transmitted from one person to another by the course of air.

mechanical and physiological explanations seem inadequate.

Psittacosis is rather rare. It is not as contagious as influenza or atypical pneumonia although it may be transmitted from man to man if there is

prolonged contact. The most frequent cause is the inhalation of the dried droppings of infected birds. The virus is apparently rather common in many kinds of birds.

This by no means exhausts the list of virus pneumonias. Many viruses that ordinarily attack other organs of the body seem capable under certain such
 cases due

to insect bites. It is likely that the pneumonic form results when the mode of infection is inhalation of an air-borne organism. Experience so far suggests that these organisms are not readily transmittable from man to man.

Pathology—The pathology of all the virus pneumonias is strikingly similar. The chief difference observed is in degree of virulence and mortality rates. Psittacosis and the influenzal types are far more virulent and have much higher mortality rates than atypical pneumonia. In other respects there is little distinction to be made.

Röntgenologic evidence points to the hilus as the area first affected. It is probable that the trachea and larger bronchi are involved at the same time. The initial effect is congestion and swelling with destruction of the ciliated columnar epithelium. The mucous membrane in the large bronchi is usually found to be intact upon microscopic examination, although there may be some ulceration. A thick, tenacious slimy secretion is sometimes found in moderate quantities.

The most severe reaction is found in the smaller bronchioles and alveoli. The process probably starts with a rather bronchiectatic-like swelling of the bronchioles. The walls in a short time become greatly thickened and the alveoli may be filled with a frank pus. Occasionally masses of fibrin will be found filling the alveoli, but ordinarily fibrin is rare. The membrane of the bronchioles is seen on microscopic examination to be desquamated as are the lining cells of the alveoli. Ulceration is common and takes place very early.

The interstitial tissue around the infected bronchioles becomes congested.

As to any anatomical divisions, the diseased areas are rather sharply delimited from healthy tissue. The distribution within a lobe is haphazard. Small areas of atelectasis or emphysema are common. There is commonly a serofibrinous fluid exuded on the visceral surfaces of the pleura and may also be found between lobes of the lung. The hilar lymph nodes become enlarged and inflamed but not purulent.

At one time secondary complications were common with a secondary infection of bacteria frequently obscuring the original process, but since it has become standard practice to administer heavy dosages of one of the

may persist for a long time. Unless however pneumonitis develops the lung will eventually completely clear itself. A chronic pneumonitis more frequently follows virus pneumonia particularly influenzal pneumonia than any other disease.

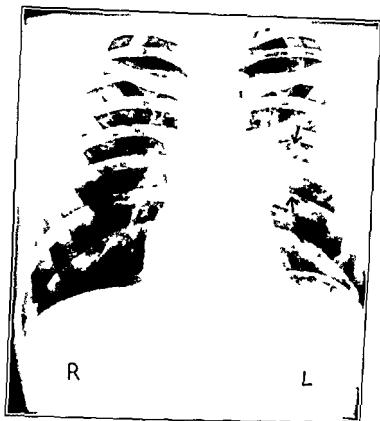


FIG 97 A twenty five year old male complaining of headache fever malaise weakness and cough for three days. Sputum did not type for pneumococci. Sulfadiazine had no response on his 100-104 spiking fever. Very excellent response to x ray therapy. Physical examination was negative throughout his entire illness. Discharged three weeks after entry.

2-6-43 Roentgenogram discloses a hazy infiltrative density in the left hilum with several hazy strands extending out from the left hilum.

Diagnosis: Virus pneumonia.

Clinical Symptoms—Although there is some differences noted in the symptomatology of the different virus pneumonias they probably are more dependent upon the severity of the attack than upon the specific virus. The forms that tend to be severer—psittacosis and influenzal pneumonia—tend also to be more abrupt in onset. However when a mild psittacosis is encountered it will be symptomatically indistinguishable from atypical pneumonia.

widely, but it is usually present at some time during the course of the disease. Chest pain is a common finding. Psittacosis may sometimes affect the patient's mental faculties. In milder instances of all virus pneumonias dizziness is sometimes found. Headache is not unusual. Occasionally abdominal pain, referred from the pleura, is described.

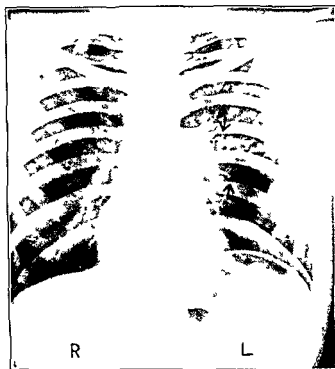


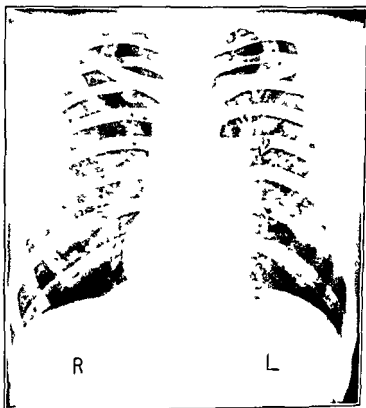
FIG. 98.—Same patient as Figure 97. Roentgenogram taken 2-10-43. The density in the left hilum has diminished and there is now hazy consolidation scattered throughout both mid lung fields.

Diagnosis: Virus pneumonia.

In army medical centers cases of atypical pneumonia have been rather frequently found by routine roentgenologic examination in patients that were and remained asymptomatic.

Physical Signs—All investigators have remarked upon the negligible physical findings in this disease. The only findings are moist or crackling. Areas of dullness may occur late in the development of the disease, if at all.

Roentgenologic Findings—The first roentgenologic evidence of virus pneumonia is a shadow near the hilus. Such hilus markings persist but irregularly scattered shadows throughout one lobe are most prominent later in the disease. The areas of infiltration are usually clearly defined. The shadows are soft as of close lying fine lines rather like a loose ball of cotton. Even when the disease is most extensive the roentgenologic shadows never achieve the homogeneity of the shadows of lobar pneumonia. There is a tendency for the scattered shadows of the early stages to coalesce



but in most instances they are patchy at the height of the illness. Occasionally small areas of increased density, representing atelectatic areas may be seen intermittently.

In about 75 per cent of the patients with atypical pneumonia the disease is unilobar. The lower lobes are most commonly involved. Influenzal pneumonia will have a primary site in one lobe but scattered involvement of other lobes is often seen.

Laboratory Findings—Normal laboratory procedures are of little aid in diagnosis of virus pneumonia. The leukocyte count usually remains normal

or slightly below normal throughout the course of the disease unless there are complications. Some leukocytosis is often found during recovery.

The use of these tests in atypical pneumonia is limited by the fact that



hospital

This roentgenogram shows a large infiltration involving the entire right upper lobe. There is also a small amount of infiltration in the right lower lobe and the left hilum.

Diagnosis: Virus pneumonia (f).

no reaction is seen ordinarily until the second week of the disease and by the failure to this date to demonstrate anything like a consistent reaction.

Differential Diagnosis The diagnosis of virus pneumonia will in many instances be suggested by the patient's history. Influenzal and atypical pneumonias both tend to be epidemic in character. Psittacosis will be suspected when a patient is found to have been around birds. Differentiation between possible kinds of virus pneumonia is to a large degree dependent upon such histories.

Virus pneumonia must be further differentiated from tuberculosis, the bacterial pneumonias and Loeffler's syndrome.

Tuberculosis — Where the patient's history is not suggestive, a mild virus pneumonia, particularly the atypical variety, may suggest tuberculosis. Differentiation cannot be made upon the basis of symptoms or roentgenologic appearances. With either condition the responsible organism may be difficult to demonstrate. Agglutination tests may indicate virus pneu-

— confirmed

— the

— This

situation occurred rather frequently in the service. Only occasionally will laboratory culture and animal inoculation be necessary. (See Fig. 8C, page 173.)

Bacterial Pneumonias — A roentgenologic examination should be sufficient in almost every instance to distinguish lobar from virus pneumonia. The denser and unbroken shadows of lobar pneumonia will seldom be mistaken for the light, patchy shadows of virus pneumonia. Broncho pneumonia, however, cannot be excluded by roentgenologic means. Differ-

Loeffler's Syndrome — This condition presents roentgenologic appearance and sometimes symptoms remarkably like those of mild virus pneumonia.

— the pro-

— tion is an

(See Fig.

101, page 200.)

BIBLIOGRAPHY

- BRETHAUER, E. A. JR. and THOMPSON, R. T. Observations of atypical pneumonias of the influenzal virus type. *Ann. Int. Med.* 20: 884, 1946.
- CRYSLER, W. I. Primary atypical pneumonia and sequestration of segmental distribution. *Am. J. Roentgenol.* 56: 324, 1946.
- EATON, M. D. Recent observations on virus pneumonia. *California & West. Med.* 63: 19, 1945.

28. EOSINOPHILIC PNEUMONIA

Eosinophilic infiltrations of the lung (Loeffler's syndrome) is because of its transient and generally symptomless nature seldom seen in ordinary practice. It is possibly much more common than its recent discovery would indicate however and it may become an important problem to diagnosis with increased use of the roentgen ray.

Etiology—Its etiology is still obscure. An allergic origin is indicated by the occasional coexistence of the syndrome and *Ascari lumbricoides* or other parasites and by the frequency with which other allergic reactions are found in patients with the syndrome. It seems most likely that Loeffler's syndrome is the product of individual reaction to a particular allergen in which the allergen varies from individual to individual.

Pathology—Because of its benign and transient nature autopsy material bearing on pathology is rare and somewhat contradictory. The material published on the subject to date confirms in general the concept of the syndrome as an allergy: there is pronounced edema of the lungs oftentimes uniquely rich in eosinophiles with fibroblasts multinucleated cells and occasionally giant cells. This fluid is often blood tinged due to the rupture of capillaries. Thrombosis of smaller vessels is common.

With the exception of the following findings, the pathology is

3

established maximum eosinophilia just after the lungs begin to clear.

Clinical Symptoms and Physical Signs—Symptoms are as remarked

tions. Physical signs are also minimal—a few moist rales and a faint dullness.

Roentgenologic Findings—The most pronounced findings are roentgenologic. They are however non-specific. The shadows may be numerous

which may be marked

Leukocytosis is moderate or
and occasionally the sedimentation rate is slightly elevated

(199)

Differential Diagnosis Diagnosis will depend upon rapidly clearing lung shadows and usually the eosinophilia (Instances of transitory pulmonary infiltrations resembling Joffler's syndrome but without eosinophilia have been reported) Examination of the patient's stools may reveal parasites For the diagnosis neoplasms fungus infection tuberculosis and virus pneumonia must be excluded Either of the first two are unlikely to present extensive shadows without pronounced symptoms All of them will be excluded by the transitoriness of the lung shadows (Differential diagnostic pictures will be found in appropriate chapters)

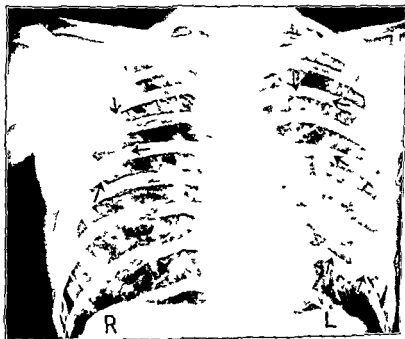


Fig 54. Eosinophilic pneumonia (Joffler's syndrome)

BIBLIOGRAPHY

- BAER A. Report of case of transient pulmonary edema (Joffler's syndrome) Ohio State M J 37 960 1941
 EMERSON J Jr Tropical eosinophilia U S Navy Med Bull 42 118 1944

1. 440, 443, 444, 445, 446, 447, 448, 449, 450, 451, 452, 453, 454, 455, 456, 457, 458, 459, 460, 461, 462, 463, 464, 465, 466, 467, 468, 469, 470, 471, 472, 473, 474, 475, 476, 477, 478, 479, 480, 481, 482, 483, 484, 485, 486, 487, 488, 489, 490, 491, 492, 493, 494, 495, 496, 497, 498, 499, 500, 501, 502, 503, 504, 505, 506, 507, 508, 509, 510, 511, 512, 513, 514, 515, 516, 517, 518, 519, 520, 521, 522, 523, 524, 525, 526, 527, 528, 529, 530, 531, 532, 533, 534, 535, 536, 537, 538, 539, 540, 541, 542, 543, 544, 545, 546, 547, 548, 549, 550, 551, 552, 553, 554, 555, 556, 557, 558, 559, 560, 561, 562, 563, 564, 565, 566, 567, 568, 569, 570, 571, 572, 573, 574, 575, 576, 577, 578, 579, 580, 581, 582, 583, 584, 585, 586, 587, 588, 589, 590, 591, 592, 593, 594, 595, 596, 597, 598, 599, 600, 601, 602, 603, 604, 605, 606, 607, 608, 609, 610, 611, 612, 613, 614, 615, 616, 617, 618, 619, 620, 621, 622, 623, 624, 625, 626, 627, 628, 629, 630, 631, 632, 633, 634, 635, 636, 637, 638, 639, 640, 641, 642, 643, 644, 645, 646, 647, 648, 649, 650, 651, 652, 653, 654, 655, 656, 657, 658, 659, 660, 661, 662, 663, 664, 665, 666, 667, 668, 669, 670, 671, 672, 673, 674, 675, 676, 677, 678, 679, 680, 681, 682, 683, 684, 685, 686, 687, 688, 689, 690, 691, 692, 693, 694, 695, 696, 697, 698, 699, 700, 701, 702, 703, 704, 705, 706, 707, 708, 709, 710, 711, 712, 713, 714, 715, 716, 717, 718, 719, 720, 721, 722, 723, 724, 725, 726, 727, 728, 729, 730, 731, 732, 733, 734, 735, 736, 737, 738, 739, 740, 741, 742, 743, 744, 745, 746, 747, 748, 749, 750, 751, 752, 753, 754, 755, 756, 757, 758, 759, 760, 761, 762, 763, 764, 765, 766, 767, 768, 769, 770, 771, 772, 773, 774, 775, 776, 777, 778, 779, 780, 781, 782, 783, 784, 785, 786, 787, 788, 789, 790, 791, 792, 793, 794, 795, 796, 797, 798, 799, 800, 801, 802, 803, 804, 805, 806, 807, 808, 809, 810, 811, 812, 813, 814, 815, 816, 817, 818, 819, 820, 821, 822, 823, 824, 825, 826, 827, 828, 829, 830, 831, 832, 833, 834, 835, 836, 837, 838, 839, 840, 841, 842, 843, 844, 845, 846, 847, 848, 849, 850, 851, 852, 853, 854, 855, 856, 857, 858, 859, 860, 861, 862, 863, 864, 865, 866, 867, 868, 869, 870, 871, 872, 873, 874, 875, 876, 877, 878, 879, 880, 881, 882, 883, 884, 885, 886, 887, 888, 889, 890, 891, 892, 893, 894, 895, 896, 897, 898, 899, 900, 901, 902, 903, 904, 905, 906, 907, 908, 909, 910, 911, 912, 913, 914, 915, 916, 917, 918, 919, 920, 921, 922, 923, 924, 925, 926, 927, 928, 929, 930, 931, 932, 933, 934, 935, 936, 937, 938, 939, 940, 941, 942, 943, 944, 945, 946, 947, 948, 949, 950, 951, 952, 953, 954, 955, 956, 957, 958, 959, 960, 961, 962, 963, 964, 965, 966, 967, 968, 969, 970, 971, 972, 973, 974, 975, 976, 977, 978, 979, 980, 981, 982, 983, 984, 985, 986, 987, 988, 989, 990, 991, 992, 993, 994, 995, 996, 997, 998, 999, 1000.

29. PULMONARY ABSCESS

THE great variety of classifications that have been attempted for pulmonary abscess is an indication of the complexity of the disease. Lung abscesses have been divided into acute or chronic depending upon an arbitrary time limit of from six to twelve weeks, they may be simple or complex

the condition will be considerably clarified if we remember that a pulmonary abscess is an inflammation within the bronchus or parenchyma of the lung in which necrosis and free purulent fluid is present and in which there is some degree of cavitation. It is not of necessity a clinical entity, and the physician's visualization of it will be clearer if he regards it as a manifestation of an inflammatory process.

However our discussion here will be limited to putrid lung abscesses. Although acute non-putrid lung abscess is probably a much commoner condition than medical literature would indicate most of these abscesses cause only mild symptoms and heal spontaneously without ever coming to the attention of a physician. Occasionally, the bacteria may invade the blood stream and cause septicemia however. When they penetrate a bronchus they usually become putrid very rapidly.

Influenzal pneumonia may be complicated by numerous small abscesses due to streptococcus hemolyticus. Roentgenologically this condition resembles miliary tuberculosis.

A serious aerobic abscess is the one which occurs in conjunction with or following bronchopneumonia and since it is essentially an extension of bronchopneumonia is discussed in that connection.

Etiology—For many years numerous investigators tried to establish a single or at least predominant etiologic process for putrid lung abscess. It is now generally agreed that infection can take place in several ways. A few case reports in medical literature point to the lymphatics as a mode of lung infection. Most lung abscesses however arise because of the

rather frequently does or where it occurs in a patient with an infected cavity it is likely that the route of infection is the direct one. A piration. Where the abscess follows an operation in some other part of the body or follows a cutaneous abscess the infection is undoubtedly carried to the lung by an infected embolus in the blood stream. It is also possible for infection and abscess to result from injury to the lung.

ag
etiologic classifications a rather sizable group. It is not necessary to seek a further means of infection for these, however since they may well

represent a long-delayed sequel to a primary infection whose micro-organisms were harbored in the lymphatics or embolism from a primary infection

A very important factor in etiological statistics is poor oral hygiene and the

widely different results. In no survey of a large number of abscesses however has a single organism been uniformly found. The various streptococci particularly the viridans and hemolyticus varieties have perhaps been most frequently found. Staphylococci are usually found in a significant number of cases in all surveys. Uniform bacilli and bacilli melan-

Where a putrid lung abscess occurs as the result of aspiration of material

of the original infection is a pathological problem. It is predicated on the basis of some clinical evidence that such abscesses are originally bacteriologically the same as the original and in many cases are non putrid but that a secondary and obscuring infection occurs shortly after such an abscess penetrates a bronchus.

An interesting sidelight to the bacteriology of lung abscess is the fact that experimentally it has been impossible to produce the abscess with any single organism.

Plainly, therefore, it is not possible to produce the abscess with any single

1
2
3
4
5

ordinarily keep the passages clear and prevent the formation of an abscess. In experimental work it has been found that abscesses only develop when some form of atelectasis occurs, thus an abscess to develop due to aspiration of contaminated material such material must partially block a bronchus. Where the mode of infection is embolism an infarct is predicated.

Putrid abscesses occur most frequently in the right lung. It is likely that there is little predilection to a particular lobe since some statistics show the upper lobe and some the lower. They are frequently multiple but the relation of multiplicity to origin aspiration or embolism is still debated. Not infrequently in abscess in one lobe will extend across a fissure and involve an adjacent lobe. A location near the pleural diaphragmatic or mediastinal surfaces of the lung is a common finding. This may be due to an embolic origin or to the fact that the infected material was very small and penetrated very deeply.

An abscess begins as an area of intense inflammation within an infarcted area or behind a blocked bronchus. Much variation has been noted in the elapsed time between the infecting process operation or other and the appearance of symptoms but usually it varies from one to two weeks. In some instances the abscess seems to begin as a group of small areas of infection that gradually coalesce.

There is usually a good deal of fibrin formation in the tissue surrounding an abscess and this may at times rather circumscribe and limit it. In exceptionally virulent instances the process of necrosis may be general with the whole lung becoming involved.

abscess will progress to the chronic condition if widespread inflammation around a slowly-enlarging abscess cavity. Chronic abscess may exist for long periods of time with only intermittent clinical symptoms.

In a small abscess of moderate virulence the fibrotic process in the surrounding tissue may be effective enough to limit the size of the abscess. Such an abscess may be present for years without seriously incapacitating the patient. In many instances the area of fibrosis will eventually calcify to some extent.

The complications possible to putrid lung abscess are numerous. The spilling-over of fluid may give rise to secondary abscesses and bronchocystitis. Because of its characteristic location on the periphery of the lung it frequently ruptures into the pleura and produces empyema. Pleuritis and adhesions are almost invariable even where there is no rupture. Metastatic infection is perhaps the most serious complication when such metastasis is in the brain it is usually fatal in a very short time unless surgical treatment is instituted promptly.

Clinical Symptoms—Instances of very rapid onset of symptoms much like those of pneumonia have been remarked upon but it is the author's experience that the development of symptoms in putrid lung abscess is ordinarily definite but slow. Prodromal symptoms are usually slight fever and malaise. These become more distinct as the abscess develops. Chills and night sweats develop. The patient's breath becomes foul and there is foul sputum in varying amounts expelled. In some instances the sudden expectoration of a large amount of foul smelling pus will indicate the rupture of the cavity into the bronchial tree. Chest pain is a usual symptom. Dyspnea is not uncommon. Hemoptysis occurs when blood vessels are eroded. Patients have bled to death when a large pulmonary vein running through an abscess cavity ruptures.

The indications of sepsis usually remain constantly present throughout the course of the disease but other symptoms may vary. When there is a sudden cessation of sputum with continued or increased sepsis the bronchial drainage has been closed. A sudden increase in the amount of fever often heralds rupture into the pleura or the development of a metastatic abscess elsewhere.

When the abscess becomes chronic there will be a progressive weakening of the patient with loss of weight and strength. Violent paroxysms of

coughing occur frequently and the sputum may be profuse and very foul. The patient may also expectorate blood. The lungs are usually rous but parts of the lung to some degree dullness is often very widespread. Atelectasis may further enlarge the areas of dullness. Rales are common. Breath sounds may be lessened or absent over a large area. In some instances the accompanying pleuritis may limit chest motion bilaterally.

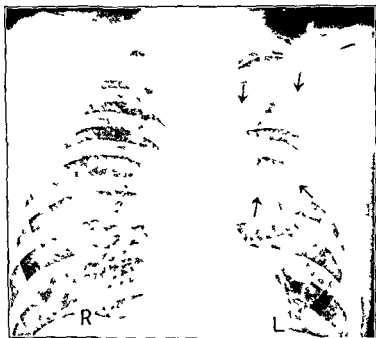


FIG. 102. A R, age forty-nine, white male.

Admitted to the hospital on July 7, 1942, with history of non-productive cough for the previous two months, though suddenly becoming productive of two to eight cups daily of foul smelling sputum ten weeks prior to entry. Chills, fever, and sweats began

Röntgen ray Findings Röntgen ray studies will be of considerable

the large shadow indicate the beginning of necrosis and liquefaction. The

typical abscess begins with several of these small areas that gradually coalesce.

In most instances of lung abscess some connection with the bronchial tree will be developed and air will enter the cavity so that a fluid level is visible. If the cavity by cough or postural drainage drains easily it may be seen on a roentgenogram as a rarified area with a low fluid level. At times there may be no fluid level at all. Ordinarily fibrosis and other encapsulating processes take place near the edge of the involved area and unless therapy or surgery intervenes the abscess cavity will grow until it



oped quite small.

The variations from this characteristic development are numerous. Occasionally no cavity is ever demonstrable roentgenologically in a patient who is found to have a large cavity at operation or autopsy. There is considerable variation possible in the thickness of the walls of a cavity

which may make interpretation of films difficult. A chronic abscess may calcify and show practically no involvement of the surrounding parenchyma. Multiple abscesses are sometimes found which are only demonstrable by body section roentgenology.

A fairly common variant is the cascade effect that may accompany a discharging abscess in which several abscesses step down from the primary one.

Empyema is a fairly common complication and may obscure the entire picture so that thoracentesis and diagnostic pneumothorax are necessary.

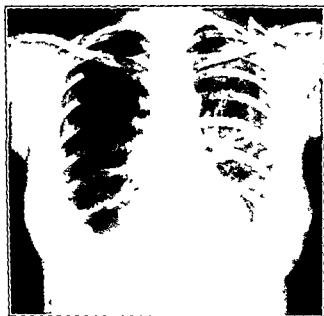


Fig. 1. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 2. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 3. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 4. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 5. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 6. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 7. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 8. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 9. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

Fig. 10. Roentgenogram of the chest showing a large, dark, irregularly shaped area in the lower right lung field, likely a pulmonary abscess.

gery somewhat. The author feels however that there are instances in which its value to diagnosis may justify its use. Where there is a large

the iodized oil will show whether or not such abscesses are connected by fistulous tracts

Bronchoscopy — Bronchoscopy has proved a useful technic in the diagnosis of pulmonary abscess by revealing the bronchus or bronchi from which the purulent material is exuded, and hence the exact areas of the lung affected.

up
cor
sor



FIG. 105 — This is a lateral view of the patient pictured in Figure 101. The interlobar effusion is unmistakable in this view.
Diagnosis: Calcified lung abscess.

Not infrequently bronchoscopy will suggest a more extensive involvement than appears in the roentgenogram.

Thoracentesis and Thoracoscopy — These technics will be undertaken only when empyema or effusion complicates an abscess.

Thoracoscopy may be used to determine the bac-

effective method of finding bacterial content than smears, but it takes longer.

The bacteria of the abscess may also be found in the blood stream. In addition the typical response to sepsis—leukocytosis, many neutrophils and high sedimentation rate—will be noted.

Differential Diagnosis — Among the primary conditions to be differentiated from abscess of the lung are emphysema, bronchiectasis and tuberculosis. These will some-

times confuse the diagnosis.

Tuberculosis — In spite of confusing roentgenologic appearances differentiation between an abscess and tuberculosis will ordinarily be suggested by the patient's history. An abscess will seldom present the drawn-out progressive development of symptoms characteristic of tuberculosis. Occasionally tuberculosis will present a large cavity with a fluid level that will



strongly suggest a putrid lung abscess. The deciding factor will be of course the demonstration of acid fast bacilli in the sputum. The two diseases occasionally coexist.

Carcinoma of the Lung — Carcinoma may undergo necrosis and clinically resemble a putrid lung abscess or it may be complicated by an abscess. The increased density of the shadow in the lung will usually be suggestive. Mediastinal lymph nodes will usually be seen to be enlarged. Bronchoscopies may provide direct vision of the tumor. When an abscess does not

respond readily to penicillin and sulfa therapy, a search for carcinoma should be made.

Bronchogenic Cysts — Ordinarily the distinct outlines of an uncomplicated cyst will not be mistaken for an abscess shadow. When a cyst becomes infected however roentgenologic distinction may be very difficult. A history of one of the etiologic factors of putrid abscess such as tonsillectomy may be elicited. Bits of cyst wall may be found in the sputum. When there are multiple cysts which is not infrequent the clearly recognized shadow of such a cyst in one part of the lung will suggest the nature



tive
Diagnosis Carcinoma of the lung producing atelectasis

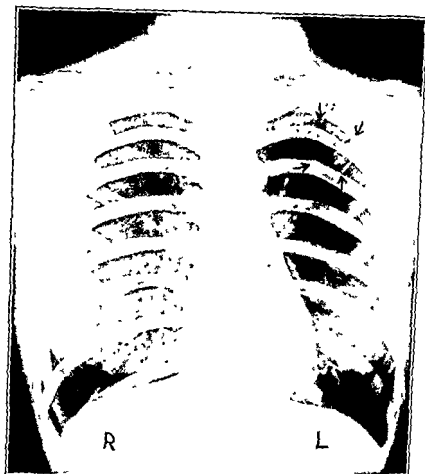
of the inflammatory lesion in another. In some instances the nature of the lesion has not been established before surgery or autopsy.

Fungus Disease — Numerous types of fungi may invade the lung and produce abscesses. The nature of the abscess when due to fungi may be suggested by the appearance of the surrounding parenchyma which will

frequently show a linear invasiveness unlike abscess of other origins. Also peculiar nodes may be seen in the area. In most instances there will be some involvement of ribs and sternum and frequently there will be skin lesions. Final differentiation depends upon the finding of the pathogenic fungi in the sputum. (See Fig. 109 page 212)



Suppurative Peribronchial Nodes—This condition in degree of sepsis, purulent sputum and so on may suggest putrid lung abscess but roentgen ray study will show the densities to be clustered around the bronchial tree—a most unusual finding in lung abscess. Roentgenologic location of the densities by the use of several positions will identify them as lymph nodes.



Coccidioidin skin test with undiluted antigen raised only 2 mm.

Tuberculin test was negative

Coccidioides immitis was cultured from her sputum and culture was confirmed by

yeast with sharp outlines under-
n peripherally. See arrows
grams show the thin walled

Bronchiectasis—Bronchiectasis may be confused with lung abscess if there are cystic dilations of the bronchi which are filled with pus. The location of the lesion in the bronchial tree will be indicative of bronchiectasis. The fluid will be discharged more easily. Roentgen ray studies after the injection of iodized oil will make differentiation certain. (See Fig 7 on page 157.)

Empyema—Symptomatically this condition closely resembles putrid lung abscess especially if a bronchial fistula develops. Differentiation can be made in most instances by a simple roentgen ray examination. However in encysted pyopneumothorax may be very difficult to distinguish without thoracentesis. Occasionally a lung abscess presents itself at the very edge of the costal area with obliteration of the pleural space. Sometimes aspiration of the cavity followed by the injection of air will indicate the location of the cavity. At other times diagnosis is not certain before operation. (See Fig 30 page 72.)

Bronchobiliary Fistula and Subphrenic Abscess—Where there is a bronchobiliary fistula or where a subphrenic abscess has ruptured through the pleura it may be difficult to differentiate the conditions from abscess of the lung. Usually a history of liver damage or a laparotomy will suggest

The examination of the sputum will often reveal micro-organisms not characteristic of lung abscess such as the entamebae histolytica. Secretions from an abdominal organ may also be at times identifiable.

BIBLIOGRAPHY

ALLEN, D. S. Etiology of abscess of the lung: experimental and clinical studies. *Arch Surg* 16: 179, 1923.

1121
 IGLAUER, S. Aspiration of blood into the larynx and trachea during thoracic surgery under general anesthesia (contribution to etiology of lung abscess). *Ann Otol Rhinol and Laryngol* 37: 231, 1928.
 KLINE, B. S. and BERGER, S. S. Pulmonary abscess and pulmonary gangrene: clinical and experimental study. *Ann Surg* 18: 491, 1923.
 1273 1922
 abscess of lung: surgical treatment.
Surg 9: 439, 1940.
 variety. *J Thoracic Surg* 12: 98.

- PILOT, I, and DAVIS, D J Studies on fusiform bacilli and spirochetes, their rôle in pulmonary abscess, gangrene and bronchiectasis, Arch Int. Med 34, 313, 1924
- ROSENBLATT, M B Putrid lung abscess, pathogenesis, prognosis, and treatment J Thoracic Surg 9, 294, 1940
- S. Experimental pulmonary abscess Arch Surg 14 221 1927
- S.
- T.
- Surg 10, 618, 1941
- VAN
- VARS 19,
- 1602 1929
- WHITEMORE, W Etiology and treatment of non tuberculous pulmonary abscess Surg Gynec and Obst 38, 461, 1924

30. PNEUMOCONIOSIS

PNEUMOCONIOSIS is a generic term now generally used to include all

dusts. Asbestosis occurs when the material inhaled is asbestos fibers. It is pathologically quite distinct from silicosis. The other recognized forms of pneumoconiosis such as siderosis, anthracosis, byssinosis, and chalcosis may be lumped together into one group.

This latter group will be discussed only incidentally because they are important only in that they may complicate diagnosis. They frequently give rise to major roentgenologic changes but they produce little or no fibrosis and do not therefore limit or interfere with lung function to any marked degree. Unlike silicosis and asbestosis they do not predicate to tuberculosis. Occasionally bronchitis or even a patchy bronchopneumonia is attributed to the action of such dusts. There is some evidence that these otherwise rather benign pneumoconioses may also lead to a higher pneumonia rate but the mechanism of such a predilection if it exists is not understood.

In the diagnosis of the clinically significant pneumoconioses, silicosis and asbestosis, a history of comparatively long exposure, usually occupational and a matter of many years is usually discoverable. Rarely will either silicosis or asbestosis be found in an individual who has been exposed to the dusts less than three years. A few situations in which silicosis developed during a period of months have been reported. Very rarely a patient with unmistakable silicosis has been encountered in which no history of significant exposure to dust has been elicitable.

Etiology—Silicosis is caused by the inhalation of particles of free silica into the alveoli of the lungs in sufficient quantity and over a sufficiently long time to produce a characteristic fibrosis. The conditions under which this occurs are complex. A feature of prime importance is the size of the silica particles. Since such particles must enter the alveoli to produce a tissue reaction it is obvious that they must be quite small. It is thought at present that particles over 5 microns in size are not pathogenic. The optimum reaction apparently occurs with particles of one micron or less in size.

Another factor in the development of silicosis is the presence of other dusts. Some dusts, particularly aluminum, seem definitely to inhibit the

demonstrated although rapidly-developing cases of silicosis have been found in the scouring powder industry where silica dust is present with alkalis.

The amount of silica inhaled is of course significant. However attempts

to pre-determine the silicosis hazard in a particular industry by silica dust count have not been especially successful. The standards that have been set up by various states and the Federal Government are based on etiologic factors such as the concentration of silica dust in the air, the duration of exposure, and the complexity of the dust. It is difficult to regard any patient exposed over a period of time to even small amounts of silicious dust as free from hazard.

As in the case of tuberculosis, the factor in

occupations in which the disease is notorious, it seldom affects more than 25 per cent of the persons exposed to the hazard. The attempts to account for this variation in individual susceptibility have not been conclusively successful. Ichmann found a high correlation between silicosis and efficient nasal passages. Mouth breathers have been indicated as particularly susceptible to the disease. Tuberculosis is certainly a predisposing factor. All these things together, however, are not enough to explain in every instance why one particular individual contracts silicosis while another equally exposed does not.

Asbestosis results from the inhalation of asbestos fibers. These fibers are usually from 10 to 200 microns long—much larger than the particles of silica dust thought to be pathogenic. The exposure time necessary for contraction of asbestosis is of course dependent upon the amount of dust present, but in general the disease seems to progress more slowly than silicosis. On the other hand, the factor of individual resistance is not so great and the percentage of people contracting asbestosis in a particular atmosphere will be greater.

Pathology. Most of the dust inhaled of any kind never reaches the lung. The shape of each nasal cavity, with two chambers connected by a narrow corridor, removes most of such dust, and the mucus secreted by the bronchial membrane is effective in catching dust that gets through the nasal passages. The ciliary action of the mucous membrane carries foreign matter that is caught by it back to the nasopharynx.

Alveolar phagocytes are responsible for the removal of such dust particles as reach the alveoli. The complete nature and activity of the phagocytes is only imperfectly understood, but it is fairly well-established that they are capable of amoeboid action. They originate in the walls of the alveoli from which they become detached and wander over the inner surface of the air sac. During this wandering they ingest particles of foreign matter with which they come in contact. Eventually they come to rest near a lymph channel opening and are taken with their load of dust into the lymphatic system.

Thus theoretically all dusts that reach the alveoli should eventually be deposited into the lymph nodes and finally into the venous blood system. In actual fact the phagocytes frequently are so overloaded with dust particles that they become lodged in some part of the lymph system and block it to a considerable extent. Also in many instances more dust is inhaled than can be removed by phagocytes; the surplus lodges in the alveoli.

Most dusts inhaled are inert and harmless, and their disposition is of

little pathologic significance. A lung black with anthracite may still function quite adequately.

Silica dust complicates this process by what is apparently chemical irritation. It is ingested by mononuclear phagocytes in the same manner as other dusts. However, it is toxic to these phagocytes. This is first demonstrated by an acceleration in their activity and a large increase in their number. Finally most of them will be destroyed by their silica load.

the phagocytes tend to accumulate even when the dust is inert. When it is silica such accumulation is more rapid. At these sites large multinuclear cells appear. The death of the phagocytes with release of their load provides a continuous stimulation to the pathologic process. Giant cells appear which closely resemble Langhans' cells of tuberculosis. Epithelioid cells and fibroblasts develop. Proliferation is rapid. Thus the typical silicotic nodule is formed with epithelioid cells in the center of the lesion and spindle-shaped fibroblasts around the periphery. Grossly the appearance of one of these nodules is onion like. It is made up of whorl like layers of scar tissue.

The degree of fibrosis in a young lesion is dependent upon the amount of silica present. The process once begun is independently continued and removal of the patient from the silica laden atmosphere will not ordinarily

the nodule is heavy. Necrosis may take place. These necrotic areas are thought to be particularly fertile areas for the growth of acid fast bacilli.

Nodulization is ordinarily discrete and takes place throughout both lungs. Its chief pathologic effect is diminished lung capacity. It is mainly important at this stage of development because of the predilection it imposes to tuberculosis. Of the patients who develop silicosis to this stage 65 to 75 per cent eventually develop tuberculosis. The percentage is somewhat less where an unusually large percentage of inert dust is also present.

Mortality directly attributable to silicosis is usually the result of massive conglomerate fibrosis. In about two-thirds of the instances encountered tuberculosis is also evident and there is some debate as to whether or not some complicating infection is not always present. The pathologic process to this stage is not well understood. It is characterized by patchy diffuse bilateral fibrosis in the lower parts of the lung superimposed upon the nodules of the earlier stage. The lesions are usually large and old nodules may be seen within it. The parenchyma affected becomes rubbery and tough and surrounding areas develop a high degree of emphysema. The condition may result from an insufficient blood supply due to constriction of arteries by nearby nodules. In postmortem examination at least the blood vessels and bronchioles are constricted and distorted. It is also possible that the fibroblasts over a long period of time result in a systemic imbalance that leads to indiscriminate production of these cells.

An acute form of silicosis is sometimes encountered in which exposure time may be as little as three months. It occurs when the silica dust particles are not filtered out by the mucous membrane.

along the lymphatic trunks and fibrosis is general and not strictly nodular. The alveolar walls become thickened. The situation is actually that of a lung overwhelmed by fine silica with the irritant properties more widely displayed than in ordinary silicosis. Acute silicosis may terminate in acute tuberculosis or in one of the pneumonias.

silicosis—up to 200 microns in length. Most foreign materials of such large size would be prevented from reaching the lung by the ciliary action of the mucous membrane, but the long narrow shape of asbestos fibers apparently makes this protective mechanism ineffective. Because of their size, however, particles do not enter the lymphatic system, which is comparatively unaffected in asbestosis.

lung in contact with the fibers results in the formation of scar tissue. The tissue seems to have two protective mechanisms available. The first is the formation of a protective layer of fibrous tissue.

serious is the appearance of fibroblasts which gradually collect along the tissue in contact with the fibers until areas of true scar tissue develop in patches along the affected bronchioles. Such development is not nodular.

The parenchyma not directly affected by the fibrotic process becomes emphysematous. The typical appearance of an asbestos lung is areas of fibrosis alternating with areas of emphysema. The two conditions may both develop in the same tissue itself will contain

The development of asbestosis to an incapacitating degree is generally slower than a similar development of silicosis. The conglomerate form of the latter disease has no counterpart in asbestosis. Asbestosis apparently continues to progress slowly. Tuberculosis is a common complication. Bronchopneumonia is probably more frequent than lobar pneumonia. Asbestosis may be acute and fulminating or chronic. There are areas of chronic infection. There

is some slight indication that asbestosis predicates to cancer of the lung

Clinical Symptoms—Both asbestosis and silicosis develop over such a

noticeable. Incapacitation is very gradual in most instances. Chest pain is characteristic of advanced silicosis or asbestosis. Where such pain is a product of the pathological condition itself, it will usually be described as tightness. If there is a concurrent pleurisy, the usually sharp pleural

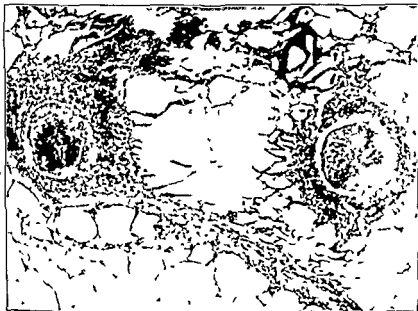


Fig. 148. Silicosis (H. E. Stain, 100x magnification).

1. 148) Diagnosis: Silicosis

pain will be noted. Frequently the patient has a cough that nothing seems to relieve. This cough will be dry and hacking if no secondary infection is present. If tuberculosis is coincident the sputum may be profuse and blood-streaked and contain acid fast bacilli. Cyanosis and clubbed fingers are common in patients with advanced asbestosis.

Physical Signs—Physical signs are mild in comparison to the extensive roentgenologic changes seen. A slight general impairment of percussion may be noted, but it is easy to overlook. Breath sounds may be decreased. Perhaps the most obvious feature is an unusual rhythm in breathing, with

a tendency to prolonged expiration. In advanced cases a decrease in chest expansion will be noted.

Roentgen ray Examination—The classification of silico is into three clinical stages: ante primary, primary and secondary. The ante primary stage is difficult even



angle

Diagnosis: Silico s grade II

Further roentgenologic examination to diagnose positively. It is particularly the first case.

nodes in the area of the hilus can be visualized. This is gradually replaced as the process advances by a mottled, "reticulo-nodular" appearance throughout the lungs but most prominent in the central areas. The primary stage is the most characteristic one. In this stage discrete nodulization is superimposed upon the mottled appearance of the ante-primary stage. These nodules first appear around the hilus of one lung usually the right. The affected area enlarges from this central location

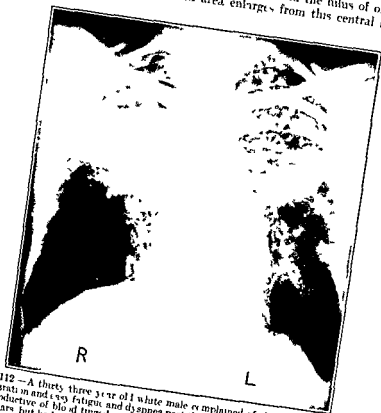


FIG. 112—A thirty-three year old white male complained of chronic cough of many years duration and easy fatigue and dyspnea past three months. Recently the cough had been productive of blood tinged sputum. He had been employed as a sandblaster for seven years but had not worked for six weeks because of his weakness. The roentgenogram of chest reveals dense consolidation in both upper lobes with dense nodulations around both hilus. Beneath the density can be seen smaller nodules sharply defined. The extreme apices and costophrenic angles are relatively clear. A radiolucent area is present in the right infraclavicular area. The sputum contained a few acid fast bacilli. (Also see Figure 83 page 166.)

Diagnosis—Silico is with tuberculous

until both lungs are rather uniformly marked by moderate-sized discrete nodules. Where the distribution is not uniform a previous infection is suggested. Unless infection is present these nodules will be of rather distinct outline. Emphysema will often completely obscure the original linear markings as the nodulization develops.

Silicosis of the secondary stage would seem roentgenologically to be the result of the fibrotic process of the earlier stages getting out of control and becoming general rather than localized. There is usually an area of massive fibrosis in each lung with areas of patchy fibrosis and large co-

When tuberculosis is present in conjunction with silicosis either precedent or antecedent nodulization will be hazier the nodules will grow more rapidly and have a greater tendency to coalesce and there will be more patchy fibrosis. Distribution of the nodules will be more uneven and cavitation will frequently be seen. Pendergrass suggests the displacement of the trachea as the best indication of whether or not coincident tuberculosis was antecedent to the silicosis displacement indicates the tuberculous process to be the earlier.

Asbestos does not show the roentgenologically distinct phases of silicosis. It is a process of diffuse fibrosis visible as thin streaks of increased density which gradually become more numerous until in the late stages it presents an appearance which has frequently been described as that of

of the lung. In a roentgenologic study of asbestos or silicosis for this matter the degree of limitation to the movement of the diaphragm is a valuable index to the degree of incipitation.

It is not unusual to find the density produced by pleuritis so extensive as to obliterate the appearances to be expected in the lower lung field.

Laboratory Studies A microscopic study of the sputum of a patient

either condition

Differential Diagnosis Perhaps the most frequent and vexatious problem in arriving at a diagnosis of either silicosis or asbestosis is the elimination of a non pathologic pneumoconiosis as a possibility. Other possibilities to be considered are fungus infection tuberculosis military malignancy sarcoidosis or advanced bronchiectasis.

Non pathologic Pneumoconioses—Legal delites as to whether a patient has silicosis or one of the non pathologic pneumoconioses particularly

be analysis of the dust to

es

ex

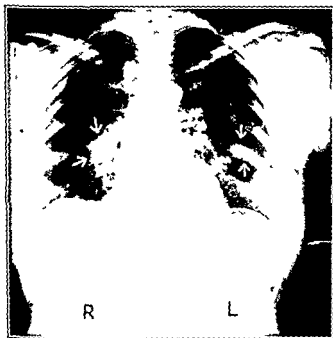
be a rather small percentage of the dust to which

worker
ica will
The

effect of the silica, roengenologically, may therefore be modified or almost totally masked by the other dust. A patient whose lungs show almost solid density due to anthracosis may or may not have an accompanying and disabling, silicosis. The distinction cannot be made roengenologically.

Tuberculosis—Miliary tuberculosis is characterized by uniform distri-

Demonstration of tubercle bacilli in the sputum might only indicate an



infected silicosis. In such an instance the patient's history is of vital importance. Careful roentgenologic studies will make differentiation possible, since the nodules of silicosis by the time that condition is spread uniformly through both lungs are much larger than the shadows of miliary tuberculosis. Also, the shadows in miliary tuberculosis are usually less distinct

kidney will produce a series of lesions in the lungs so widespread as to resemble silicosis. Such lesions will however cast soft indistinct shadows rather like a silico tuberculosis. Where the appearances are of a silico tuberculosis but no acid fast bacilli are demonstrable the possibility of metastatic malignancy must be ruled out by microscopic examination of the sputum for malignant cells and by a thorough examination for a primary growth.

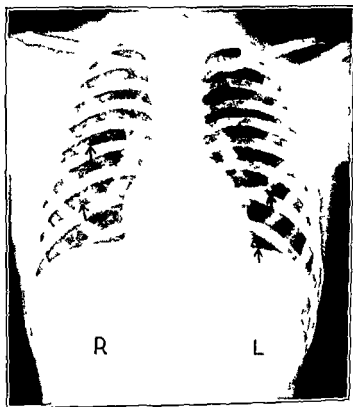


FIG. 114.—R. I. eight year old colored male. The patient entered hospital with a cough.

..

Fungus Infection—Histoplasmosis and blastomycosis are most apt to

Occasionally a patient is encountered with sharply outlined rather uniformly distributed shadows in the lung that are virtually indistinguishable from the shadows of silicosis yet the patient will be completely asymptomatic. Roentgen ray examinations over a period of time will show no change in size of these nodules. Recent studies indicate that these shadows are due to healed and calcified lesions due to histoplasmosis infection.

BIBLIOGRAPHY

- BOHRER M G Pulmonary siderosis two cases with reticulo-endothelial siderosis Arch Path 10 179 1930
- COOKE W I Industrial diseases of lungs with special reference to silicosis Practitioner 137 313 1936
- DREFASEN W C and JONES R R Anthracosis J A M A 107 1173 1936
- DUNN I and BIGNALL D J T Graphic pneumoconiosis compiled by cavity
cunoconiosis in dockers dealing
radon Pub Health Bull 187
was 1411 D C pp 155 157 1943
- 101 584 1933
- PA
- SA
- SA
- SH
- SI
- 1939
- The Health of workers in dusty trades Pub Health Bull 187 Washington D C 1929
p 165
- WILLIS H S Pneumoconiosis and tuberculosis Medicine 9 413 1930
- WOOD W B and GLOVE S R Pulmonary asbestosis review of 100 cases Lancet
2 1383 1934

31. PULMONARY MYCOSIS

The emphasis in the present interest in fungus disease is upon the individual fungus rather than upon the disease.

It is necessary to re-

The various fung-

ities and early di-

consider mycosis a possibility in any obscure lung disorder

Since many of the pathologic fungi are endemic or have an occupational etiology, much has been made of the importance of occupational and res-

Mycology Fungi are simple organisms that reproduce by sporation. They are differentiated from algae in that they lack chlorophyll and thus are unable to manufacture their own food but must live on the organic matter of a host. In many instances the natural host of the fungi pathogenic to man has not been determined but it is possible that even in the e-

Many fungi ordinarily considered non pathogenic may produce lesions in rare instances when conditions are especially favorable. The fungi most likely to infect the chest are *Histoplasma capsulatum*, *Cryptococcus neoformans*, *Coccidioides immitis*, *Candida albicans*, *Blastomyces* and *Sporotrichum*. Schenck among the yeast like fungi, the *Aspergilli* among the mold like fungi and the *Aetnomyces* species among the higher bacterial forms.

HISTOPLASMOSIS is caused by an organism that may be seen microscopically in fresh sputum as round or oval cells of from 2 to 4 microns in diameter. The capsules are thin. A single granule is sometimes seen in the interior of the cytoplasm. Reproduction is by budding with only one bud attached at a time.

Instances of histoplasmosis have been widely reported. Until quite recently, the disease was considered a variably fulminating mass roentgen ray ever suggest that a mild and self resolving form of the disease is endemic and wide-spread in that area.

MONILIASIS is

Candida albicans

apparently the on-

to man. In fresh

as round, oval or elongated cells 2 to 5 microns in diameter. Budding

forms and mycelium are often observed. The cells tend to group themselves in pairs or small clusters.

No particular climatic or geographic predilection by this fungus has been determined. It is a common inhabitant of the mucous membranes and skins of healthy individuals. Most instances of infection have occurred in debilitated adults and in children, so bodily resistance is obviously an important factor in the development of mycosis. Moniliasis is the only

Blastomyces dermatitidis is a fungus in diameter. Its capsule is double-contoured and highly refractive. Reproduction is by budding. Although other types of *Blastomycetes* are also found.

Coccidioides immitis affects both man and animals. The disease in which the lungs are involved seems chiefly to be contracted by people living in a damp and unhygienic environment. *Coccidioides immitis* is caused by *Cryptococcus neoformans*, which has also been called *Torula histolytica*. This organism when seen microscopically in fresh sputum closely resembles the organism of blastomycosis, except that *Cryptococcus* is not so refractile and does not show the pronounced double contours of the *Blastomyces*.

Coccidioidomycosis is caused by the *Coccidioides immitis*. Microscopically this fungus is seen in sputum as a spherical double-contoured organism ranging in diameter from 5 to 75 microns. Multiplication is by endospore and a characteristic feature of the organism is the speckled appearance of its interior due to the endospores. At the climatic state of development the parent organism ruptures and the endospores are released to recommence their life-cycle.

The usual host for *Coccidioides immitis* is dry decaying vegetation and transmission to man is apparently by air borne dust. This parasite flourishes in a dry climate and it is endemic in the San Joaquin Valley of California and in Arizona, New Mexico and West Texas. A few instances have been reported elsewhere.

Sporotrichosis is caused by *Sporotrichum Schenckii*. In fresh sputum these organisms may be seen as oval bodies from 2 to 5 microns in length. The capsules are thin. They may be found singly or in small clusters. Reproduction is by budding.

Instances of sporotrichosis have been reported from many countries but it is most common in the North Central States of the United States and in France. The primary hosts for the fungus are leaved plants. Barberry has been particularly indicated as a source of infection. The fungus may also attack animals and in some instances animals seem to act as mechanical carriers.

Aspergillosis may be caused by several species of *Aspergillus* but *Aspergillus fumigatus* is probably most pathogenic for man. They are seen in fresh sputum as round or slightly oval bodies. They are smaller than the

the fungus. It is also a rather frequent secondary invader.

ACTINOMYCOSIS is caused by the anaerobic *Actinomyces bovis* or one of several species of aerobic *Nocardia*. They may be detected without a microscope as small sulphur granules that upon microscopic examination are seen to be composed of mycelial filaments. The filaments around the edges of these granules are arranged radially with their outer ends clubbed.

Distribution of instances of actinomycosis is world wide. The anaerobic *Actinomyces bovis* has been found around the tonsils and decayed teeth of apparently normal individuals suggesting that most cases of actinomycosis are endogenous. The various species of *Nocardia* have been found in the soil.

STREPTOTHRICOSIS is classed by many authorities as a form of actino-

Due to the confusion over nomenclature and the difficulty in distinguishing

from the inhalation of air borne spores. Moniliasis and actinomycosis usually infect the lungs by extension from a primary focus in the mucous membrane of the trachea or larger bronchi. Blastomycosis ordinarily reaches the lung in a process of generalized systemic infection. Any of the more virulent fungi can be systemic. When the primary focus is cutaneous a history of trauma can often be elicited although penetration of healthy skin by fungi is apparently possible. A fungus infection may re-

Since body mechanisms for resistance to disease are often important fungi the physical condition of factor in the contraction of a my-

coses are typically granulotrophonuclear cells and fungi. The smaller micro-organisms

will frequently be seen to be engulfed by macrophages or giant cells. Surrounding the zone of necrosis there will be an area of granulation tissue. Cavitation is common but seldom extensive.

Where the infection of the lungs is by the blood stream the lesions may be military in character. At other times they are patchy and bronchopneumonic in character. Any part of the lung may be invaded but ordinarily the lesions are hilar and basal except where the lungs are infected by direct extension from the trachea or larger bronchi the original lesions are found in the bronchioles and smaller bronchi. The alveoli are secondarily affected.

The pus from mycotic lesions tends to burrow through tissue. Actinomycosis is particularly apt to be characterized by skin sinuses but may penetrate any of the thoracic organs. All the fungi tend to develop connections between cavities and necrotic areas. Empysemata and abscesses develop as areas of the lung become partially or totally occluded.

The blood manufactures specific antibodies to resist the action of the different fungi in the same way it does for pathogenic bacteria. In infections of low virulence particularly when the infection is coccidioidomycosis or histoplasmosis the fungi is usually resisted successfully with eventual calcification of the lung lesion. The genus of the fungi is not however an accurate measure of virulence and these ordinarily milder mycoses have fulminating forms. Where the infection is highly virulent and extending rapidly there may be no antibodies in the blood.

All of the mycoses have some tendency to metastasis and the more virulent the infection the more widespread its manifestations are apt to be. Blastomycosis sometimes involves almost every organ of the body. Actinomycosis is particularly apt to involve the bones especially the ribs, scapula and sternum. Metastasis to the brain is common.

Clinical Symptoms—The symptoms of fungus disease of the lung are in general those of any inflammatory infection. The typical symptoms are fever, sweats, chills and cough. The degree of severity of these symptoms is in the case of those infections with both a benign and acute form a measure of the severity of the infection. A most significant symptom is distributed muscular pain like that characteristic of influenza. Symptoms are usually fairly constantly present although there may be irregular remission of fever in severe infections. Manifestations in its milder form is apt to be remittant. Quantities of non purulent sputum are usually raised only after other symptoms have been present for several days. Marked hemorrhage of the lung is rare in most of the mycoses although Jacobson says it is common in aspergillosis. The sputum may become streaked with blood as the lesions progress. The evidence recently uncovered in some of the Middle Western states of widespread sub-clinical histoplasmosis emphasizes the great variation in severity of symptoms and manifestations that exist in fungus diseases. In some instances the possibility of mycosis has been suggested on the basis of roentgenologic changes all out of proportion to the mildness of the symptoms.

Physical Signs—The physical signs of mycosis will depend upon the nature and amount of the involvement. Dullness on percussion, râles and increased breath and whisper sounds are the most common findings. Diminished expansion of the chest due to fibrinous pleurisy is quite common.

observed. In general, the physical signs will not indicate as severe and as extensive a process as roentgenograms would suggest.

Some of the mycoses present diagnostically significant cutaneous or subcutaneous lesions in four fifths of the cases encountered. A complete phys-



FIG 11a S C age thirty three white female saleslady
The patient entered hospital with history of afternoon fever, malaise, and night sweats

Roentgen-ray Findings — Diagnosis of fungus disease cannot be made upon the basis of roentgenologic examination alone, but in many instances an unusual combination of shadows may suggest mycosis to the examiner.

All the pulmonary mycoses tend to be marked by unusual hilar thickening and are usually seen roentgenologically as single, irregular and enlarging



FIG. 116 — This is a lateral view of the same patient as in Figure 11a. Radio translucent area outlined by arrow.

Diagnosis — Pulmonary coccidioidomycosis.

shadows, ordinarily unlobular. Spread is from the hilus. These higher fungi are the only ones that may produce marked and extensive fibrosis.

The other mycoses are even more diverse in their roentgenologic appearances. The most typical appearance is of rather patchy and irregular shadows emanating from the hilus. Such shadows indicate involvement of the parenchyma. Lung abscesses are not unusual, but they are seldom

observed. In general, the physical signs will not indicate as severe and as extensive a process as roentgenograms would suggest.

Some of the mycoses present diagnostically significant cutaneous or subcutaneous lesions in four-fifths of the cases encountered. A complete physical examination will often lead to the diagnosis of an otherwise obscure chest infection. The examiner should particularly watch for irregular nodulation along lymph channels—a very common manifestation of coccidiosis



Roentgen ray Findings—Diagnosis of fungus disease cannot be made upon the basis of roentgenologic examination alone but in many instances an unusual combination of shadows may suggest mycosis to the examiner.

All the pulmonary mycoses tend to be marked by unusual hilar thick-
and are usually seen roentgenologically as single irregular and enlarging



FIG. 116 This is a lateral view of the same patient as in Figure 115. Radio-transparent area outlined by arrow.
Diagnosis: Pulmonary, coccidioidomycosis.

shadows ordinarily unilobular. Spread is from the hilus. These higher fungi are the only ones that may produce marked and extensive fibrosis.

The other mycoses are even more diverse in their roentgenologic appearances. The most typical appearance is of rather patchy and irregular shadows emanating from the hilus. Such shadows indicate involvement of the parenchyma. Lung abscesses are not unusual but they are seldom

observed. In general, the physical signs will not indicate as severe and as extensive a process as roentgenograms would suggest.

Some of the mycoses present diagnostically significant cutaneous or subcutaneous lesions in four-fifths of the cases encountered. A complete physical examination will often lead to the diagnosis of an otherwise obscure chest infection. The examiner should particularly watch for irregular nodulation along lymph channels—a very common manifestation of coccidiosis.



FIG. 115.—S. C., age thirty three, white female saleslady. The patient entered hospital with history of afternoon fever, malaise, and night sweats.

Roentgen ray Findings—Diagnosis of fungus disease cannot be made on the basis of roentgenologic examination alone but in many instances unusual combination of shadows may suggest mycosis to the examiner. All the pulmonary mycoses tend to be marked by unusual hilar thick-

nesses. They are usually seen roentgenologically as single irregular and enlarging



FIG. 116.—This is a lateral view of the same patient as in Figure 115. Radio-translucent areas outlined by arrow.

Diagnosis—Pulmonary coccidioidomycosis.

usually ordinary unilobular. Spread is from the hilus. The higher lobes are the only ones that may produce marked and extensive fibrosis.

The other mycoses are even more diverse in their roentgenologic appearances. The most typical appearance is of rather patchy and irregular shadows emanating from the hilus. Such shadows indicate involvement of the parenchyma. Lung abscesses are not unusual but they are seldom

ol
ex

cutaneous lesions in four fifths of the cases encountered. A complete physical examination will often lead to the diagnosis of an otherwise obscure chest infection. The examiner should particularly watch for irregular nodulation along lymph channels—a very common manifestation of coccidiosis.

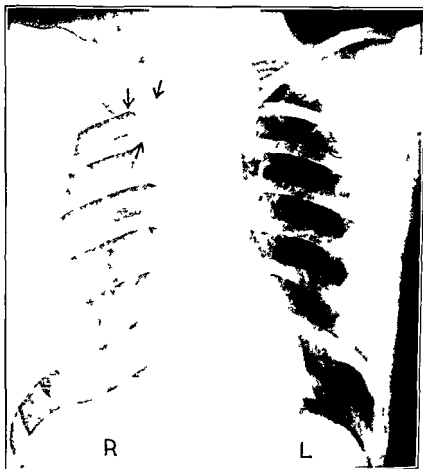


FIG. 11a.—S. C., age thirty-three, white female saleslady. The patient entered hospital with a history of afternoon fever, malaise, and night sweats. There was no loss of weight.

Roentgen-ray Findings—Diagnosis of fungus disease cannot be made upon the basis of roentgenologic examination alone, but in many instances an unusual combination of shadows may suggest mycosis to the examiner.

All the pulmonary mycoses tend to be marked by unusual hilar thickening and are usually seen roentgenologically as single, irregular and enlarging



FIG. 116.—This is a lateral view of the same patient as in Figure 115. Radio translucent area outlined by arrow.

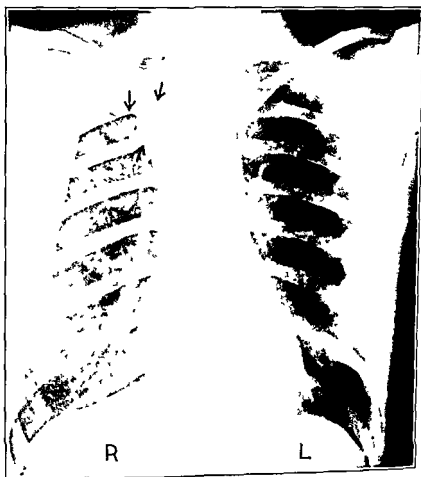
Diagnosis—Pulmonary coccidioidomycosis.

shadows, ordinarily unilobular. Spread is from the hilus. These higher fungi are the only ones that may produce marked and extensive fibrosis.

The other mycoses are even more diverse in their roentgenologic appearances. The most typical appearance is of rather patchy and irregular shadows emanating from the hilus. Such shadows indicate involvement of the parenchyma. Lung abscesses are not unusual, but they are seldom

observed. In general, the physical signs will not indicate as severe and as extensive a process as roentgenograms would suggest.

Some of the mycoses present diagnostically significant cutaneous or subcutaneous lesions in four-fifths of the cases encountered. A complete physical examination will often lead to the diagnosis of an otherwise obscure chest infection. The examiner should particularly watch for irregular nodulation along lymph channels—a very common manifestation of coccidioidosis.



Röntgen ray Findings — Diagnosis of fungus disease cannot be made upon the basis of roentgenologic examination alone but in many instances an unusual combination of shadows may suggest mycosis to the examiner. All the pulmonary mycoses tend to be marked by unusual hilar thickening. In most instances such thickening plus an accentuation of normal lung markings is the only roentgenologic evidence of moniliasis. Actinomycosis and streptothricosis tend to spread by direct extension and are usually seen roentgenologically as single irregular and enlarging



Fig. 116. Lateral view of the same patient as in Fig. 115. Pulmonary mycosis indicated by arrow.

shadows ordinarily unilateral. Spread is from the hilus. These higher fungi are the only ones that may produce marked and extensive fibrous masses. The other mycoses are even more diverse in their roentgenologic appearances. The most typical appearance is of rather patchy and irregular shadows emanating from the hilus. Such shadows indicate involvement of the parenchyma. Lung abscesses are not unusual but they are seldom

large, and it is rare to find them multiple. One or two in a "bronchopneumonic" shadow is a usual finding. They are usually thin-walled. Drainage of such cavities is not established readily, and so they are usually filled with pus and necrotic debris.

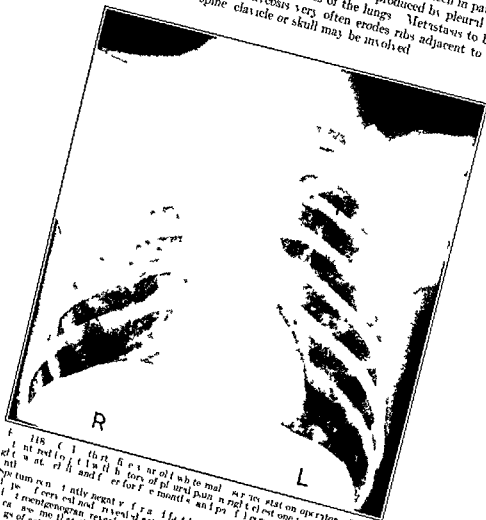
1



FIG. 117.—Tuberculosis.

Coccidioides immitis skin test with undiluted Smith antigen was strongly positive.
Tuberculin test was negative.
Coccidioides immitis was cultured from her sputum and culture was confirmed by
shadow indicated by the ar-

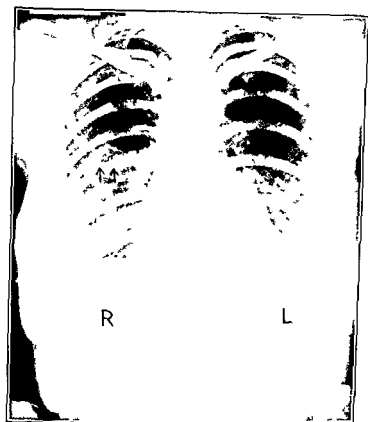
general the lesions of fungus disease tend to be more cloudy than the shadows of other conditions. There is a marked predilection for the lower lobes and hilus although apical involvement does not rule out mycosis. Pleural thickening and empyema pockets are frequently seen in patients with advanced mycosis. Sometimes the shadow produced by pleural fluid is all completely obscure the lower lobes of the lungs. Metastasis to bone is quite common. Actinomycosis very often erodes ribs adjacent to the lung lesion. The spine, clavicle or skull may be involved.



118. (1) Chest film is all white mal. Artery station operator. The
 t. t. nt red to t. l. w. h. t. o. r. of pl. ur. l. p. n. n. g. l. t. e. l. t. o. n. e. s. a. r. p. t. s. to. n. t. r. s.
 e. l. t. u. s. t. e. l. f. i. and f. e. r. f. e. m. o. n. t. h. s. a. n. l. p. a. f. l. e. r. s. a. l. n. o. p. a. t. l. y. f. o. r. t. h. e. s.
 n. n. t. l.
 s. p. e. t. u. m. r. e. n. t. a. t. l. y. n. e. g. a. t. i. v. e. f. o. r. a. f. a. t. b. a. r. i. i.
 i. l. p. e. f. e. r. s. e. a. l. n. o. d. r. e. v. e. a. l. a. c. t. i. n. o. m. y. c. o. s. i. s.
 (1) i. s. o. e. n. o. g. r. a. m. r. e. v. e. a. l. s. n. i. l. t. r. a. t. i. o. n. o. f. t. h. e. u. p. p. e. r. o. n. e. t. h. r. d. o. f. t. h. e. r. i. g. h. t. l. u. n. g. f. i. l. l.
 O. n. c. a. s. e. m. e. t. a. t. a. t. e. c. o. n. d. i. t. i. o. n. i. n. t. h. e. l. u. n. g. i. s. d. u. e. t. o. a. c. t. i. n. o. m. y. c. o. s. i. s. b. e. c. a. u. s. e. o. f. t. h. e.
 f. i. l. l. i. n. g. o. f. a. c. t. i. n. o. m. y. c. o. s. i. s. i. n. t. h. e. c. e. r. v. i. c. a. l. l. o. p. e. s.
 i. s. a. g. n. a. Pulmonary actinomycosis

Laboratory Tests—Positive diagnosis of a mycosis requires laboratory

work made only by culture or animal inoculation, a procedure undertaken only in well-equipped laboratories



Some care must be taken in diagnosis on the basis of laboratory findings. The frequent presence of aspergillus in the laboratory as a contaminant may easily mislead. *Actinomyces* and *Candida albicans* may be present in oral cavities and sputum without being pathogenic. In some instances bronchoscopic aspiration directly from infected bronchi is advisable. A diagnosis of mycosis is justified only when pathogenic fungi have been found in the sputum repeatedly.

A further consideration is that fungi may invade secondary and disguise the primary lesion and that a fungus lesion can be disguised by secondary bacteria. Diagnosis must be made with caution. Complement-fixation tests have been developed for all the important fungi and can be of considerable help in diagnosis, although a negative response, particularly in case of a fulminating condition, is not conclusive. **Differential Diagnosis**—The protean manifestations of the mycoses make of course, for a large number of possible confusions in diagnosis.



FIG. 120.—T.M. series of four-year-old white male had pertussis and upper respiratory infection and dry cough. Chest roentgenogram revealed an early infiltrative density in the right lower lobe which remained unchanged over period of ten days. Bronchoscopy revealed an early obstructing lesion in the right lower lobe bronchus with slight secondary inflammatory changes and cancer cells. Lobectomy revealed a bronchogenic cancer of squamous cell type. Roentgenograms in the P.A. and right lateral projection disclose a hazy infiltrative extension into the medial right posterior segment of the right lower lobe with slight flattening of the right costophrenic angle. The remainder of the chest is essentially negative.

Diagnosis—Carcinoma of the lung producing atelectasis. They have most frequently been mistaken for tuberculosis but carcinoma lung abscess, bronchitis, bronchiectasis and the pneumonias must also be considered. **Tuberculosis**—Although tuberculous is usually apical and the mycoses are usually basal and hilar, neither predilection is invariable enough to be

reliable diagnostically. Diagnosis can only be made by the demonstration of one organism or another in the sputum but there will often be indications of one or another condition. *Tuberculosis does not present the pronounced*

hilar

the

with

larger and more numerous cavities. None of the fungi are apt to present the extensive fibrosis of tuberculosis. Mycosis will frequently be indicated by cutaneous or osseous metastasis. (See Fig 136 page 275)

Carcinoma—Roentgenograms will show densities more homogeneous in malignancy than the densities associated with mycoses ordinarily and if they are taken serially they may show these densities gradually enlarging. However in early malignancy a roentgen ray study is not an infallible basis of distinction.

ight

lem

35)

ab

penicillin or sulfa therapy. Mycosis may be suspected when parenchymal involvement is vaguely defined and when hilar thickening and lymph node enlargement is pronounced. History is important to diagnosis. (See Fig 103 page 206)

ever bronchitis progresses in spite of modern therapy a search for fungi should be undertaken.

Bronchiectasis—Fungi will not ordinarily mimic the classic roentgenologic appearances of bronchiectasis since it tends to erode the parenchyma to a greater extent. However a fungus can easily be superimposed upon an already established bronchiectasis. Such a condition can only be diagnosed by finding the organism in the sputum repeatedly. (See Fig 75 page 157)

Pneumonia—Fungus disease is not apt to be mistaken for lobar pneumonia but it has been diagnosed as bronchopneumonia or as atypical pneumonia. Persistence of symptoms beyond any normal expectation for pneumonia will suggest mycosis. Care must be used in diagnosis since the organisms of either bronchopneumonia or mycosis may be found in the

BIBLIOGRAPHY

- CARTER R. A. Cocci of the granuloma roentgen diagnosis. *Am. J. Roentgenol.* 25: 715 1931
 ——— Pulmonary mycotic infections. *Rad.ology* 26: 551 1936

JUD
KAY

KISHI

- REPORT ON PROGRESS ACT. ACT. TUB. 19, 1943, 1212
- KARLSON J. M. The isolation and identification of pathogenic fungi from sputum. *Am Rev Tuberc* 46, 363 1942
- The isolation and identification of pathogenic fungi from sputum. II, *Am Rev Tuberc* 55, 395 1947
- MARTIN, D. S. Complement fixation in Blastomycosis, *J Infectious Dis* 57, 291 1935
- MARTIN, D. S. and SMITH D. J. Laboratory diagnosis of Blastomycosis. *J Lab & Clin Med* 21, 1289 1936
- Blastomycosis (American Blastomycosis, Gilchrist's disease), review of literature. *Am Rev Tuberc* 39, 275 1939
- Blastomycosis (American Blastomycosis, Gilchrist's disease) a report of thirteen new cases. *Am Rev Tuberc* 39, 488 1939
- MEHREZ G. and MICKENFELT, R. S. Identification of a streptothrix isolated from human being infected with it, *J Infect Dis* 47, 425, 1930
- MELENDY, H. E. Pulmonary histoplasmosis, report of 2 cases. *Am Rev Tuberc* 44, 210 1941
- PAUMER C. I. Nontuberculous pulmonary calcification and sensitivity to histoplasmin. *Pub Health Rep* 60, 513 1945

CLINICAL TRAINING
Nurses training in

In 14, 233 1935
Tuberc 28, 438

1135

- SINGER, J. J. and BALLON H. C. Streptothricosis, *Am Rev Tuberc* 22 233, 1930
- SMITH D. T. Diagnosis and treatment of common fungous infections of the lungs. *Tr Tub Annul Meet Nat Tuberc Assoc* 1933
- On histomycosis of the lungs, report of a case due to species of *Geotrichum*. *J The Path Soc* 3, 241 1934
- TORFEL, I. Fungoid disease of the lungs. *Acta Tuberc Scandinar* 20, 212 1946
- Threshold lung fungoid disease resembling tuberculosis or Morbus Schussmann. *Acta med Scandinar* 125, 191 1946
- WANDERSTEEN O. H. Role of surgery in treatment of actinomycosis, *Ann Surg* 104, 732 1936

32. HODGKIN'S DISEASE (HODGKIN'S LYMPHOMA)

HODGKIN'S disease is a generalized condition affecting at times almost all parts of the body. In perhaps three-fourths of the instances encountered the mediastinal lymph nodes are affected and about one-fourth of the time the disease presents pulmonary lesions.

Because of the confusion that exists at the present time in the definition

concept of neoplastic origin has been discarded by most authorities and

basis of the inflammatory nature of the tissue changes to seek a virus or bacterium as agent. To date no such agent has been established.

Pathology In a great majority of cases evidence of Hodgkin's disease is first noted in the cervical lymph nodes. Autopsy findings however indicate that the disease may originate in other nodes with some frequency.

There is an increase in lymphocytes and characteristic development is an accumulation of cells which may become greatly enlarged.

main discrete

Fibrosis appears very early and may replace other structures rapidly.

Commonly the visceral organs are involved by extension of the disease through lymphatic channels. Rarely rupture of the lymph node capsule takes place. The lungs, spleen and kidneys are most frequently invaded. The most common pulmonary manifestation is nodular.

When visceral organs are involved the disease may spread to the blood and bone marrow and to the visceral organs is other

In rare instances Hodgkin's disease is terminated by a true lymphoma.

quiescence may obtain for years but no definite cure has been reported.

Clinical Symptoms —Hodgkin's disease frequently comes to the attention of the physician solely because of enlarged lymph nodes there are no other symptoms. In the paraneoplasia the patient is ordinarily in apparent good health and unless the hypertrophied nodes impinge upon nerves there is no pain.

Symptoms develop with the transition to granuloma. In a few instances the nodes become painful. There is loss of weight and strength and the

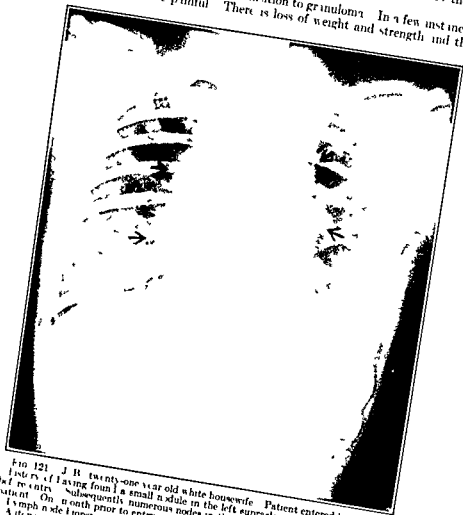


FIG. 121. J. R., twenty-one year old white housewife. Patient entered hospital with history of having found a small nodule in the left supraclavicular area eight months before entry. Subsequently numerous nodes in the axilla and neck were noted by the patient. On month prior to entry patient became increasingly dyspneic. Lymph node biopsy revealed Hodgkin's Disease with widespread lymphogranulomatosis. A chest roentgenogram revealed a large homogeneous mass with irregular edges occupying the mediastinal area outlined by arrows. See also Figure 31, page 75.

Diagnosis: Hodgkin's Disease

patient becomes listless. The jagged Pel-Ebstein fever chart in which intervals of normal temperature gradually become shorter is most significant. A generalized pruritis is very commonly noted.

Physical Signs—Superficial lymph nodes can often be palpated when they are not visible. In Hodgkin's granuloma the spleen and sometimes the liver can be palpated. The abdomen may feel spongy and be swollen.

There are seldom any physical signs to be elicited from examination of the chest itself. In rare instances the lymph nodes impinge upon a bronchus so as to produce atelectasis or emphysema. The usual physical signs of these secondary conditions may be noted.

Roentgen ray Findings—The most characteristic finding in a roentgeno-

involved but almost always the process is much more advanced on one side than on the other. There is seldom pronounced mediastinal displacement.

This occasionally produces atelectasis with subsequent bronchiectasis. Cavitation probably indicates a coexistent infection such as tuberculosis although cavities have been found where other infection was not demonstrable.

Laboratory Tests—Analysis of the blood is of little diagnostic value. Anemia to some degree is a very common finding in Hodgkin's granuloma but it is a tendency and not a diagnostic criterion. Otherwise the blood findings may be extremely variable.

Diagnosis depends upon the recognition of characteristic tissue changes in a biopsy specimen. A lymph node of moderate size not previously in-

Pathology

Occasionally biopsy of the skin when pruritis is present may show typical Dorothy Reed cells.

and tuberculosis.

Lymphosarcoma may be suggested by hardness of palpated nodes and tenderness but differentiation is positive only upon the basis of biopsy studies. Treatment and prognosis of the two conditions are essentially the same. (See Fig 51 page 108.)

Chronic Lymphatic Leukemia—This disease will ordinarily be indicated

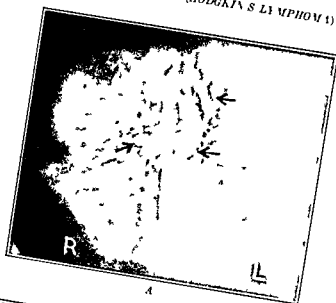


FIG. 109. Radiogram of excised lungs. The inner third of the right lung field shows diffuse mottling and the inner half of the left lung field shows diffuse mottling. There are also arrows pointing to the hilar regions in both lungs.

FIG. 110. Radiogram of excised lungs. The inner third of the right lung field shows diffuse mottling and the inner half of the left lung field shows diffuse mottling. There are also arrows pointing to the hilar regions in both lungs.

Diagnosis: Bilateral hilar lymphadenopathy and bilateral hilar lymphadenopathy. Tuberculosis of the hilar lymph nodes with bronchopneumonia. (From Quarterly J. of Med. vol. 29 April 1952)

by unusually high lymphocyte counts in analysis of the blood. The enlarged lymph nodes are apt to be fused together and present microscopically a uniform lymph structure.

Tuberculosis—Tuberculosis of the lymph nodes may be extremely difficult to distinguish from Hodgkin's disease. Both present caseating necrosis. Tuberculosis should be suspected when single nodes are involved and when the skin over the nodes is inflamed. Hodgkin's disease reacts more readily to irradiation. Differential diagnosis depends upon laboratory methods—the demonstration of acid fast bacilli or Dorothy Reed cells in the lesion. The diseases do not infrequently coexist. (See Fig. 134, page 273.)

BIBLIOGRAPHY

BELL, E. T. Textbook of Pathology, 6th Ed. Philadelphia: J. B. Lippincott & Co. 1947, p. 338.

CRAYER, L. F., BRUND, R. R. and TYLER, H. V. Lesions of lungs in lymphomatous diseases. *Am J Roentgenol* 45: 342, 1941.

COHEN, S. J. and COHEN, S. J. The Diseases of the Chest.

1944

Am J Med 23: 37, 1946
roentgenological

Am J M

J A M A 113

TURNER, J. C., JACKSON, H. JR. and PARKER, F. JR. Histologic relation of eosinophils to Gordon phenomenon in Hodgkin's disease. *Am J M Sc* 195: 27, 1938.

33. PULMONARY EMPHYSEMA

THERE are really only two distinct forms of emphysema, interstitial in which air collects in tissues which do not normally contain gas and pulmonary in which the alveoli become chronically distended and in which there is temporary or permanent loss of elasticity in the alveolar walls. The second type is the subject of this chapter. Further subdivision within this group is frequently undertaken but its impracticability is indicated by the variation found in such division and in definition of the subdivisions. Compensatory emphysema constitutes a rather distinct clinical entity, but its distinctive characteristics are mere use of elasticity and regression when the usual condition is removed. These characteristics are seldom found in other forms of emphysema.

Etiology—Either of the two distinguishing characteristics of alveolar emphysema may be precedent. In atrophic emphysema the cause is deterioration of alveolar walls apparently due to insufficient blood supply. Starvation of the tissue produces flabbiness—which is over-distention. However chronic over-distention is the more important cause in most instances of emphysema. Such emphysema ordinarily develops over a rather long period of time in the presence of some other chronic condition. The pathologic factor is a vital factor. Thus in far the greatest number of walls the chronicity is a vital factor. Thus in far the greatest number of instances of pathologic emphysema the etiologic factor is interference with the free passage of air in the bronchi by tumor or foreign body or by swelling of the bronchial lumen itself due to asthma or infection. Where there is not unimpeded air flow the alveolar walls are strained by the partial vacuum that the depression of the diaphragm and the elevation of the ribs tends to create upon inhalation since an abnormal pressure gradient is created. Where the air does not flow freely it will also be seen that the patient will normally unused expiratory muscles into play for exhalation and thus compresses the air contained in the obstructed portion of the lung. Thus the alveolar walls are rather constantly subjected to excessive pressures. The first factor the delayed filling of the lung upon inspiration is undoubtedly the most important as evidenced by the usual obstruction in maximum emphysema at the pleural and diaphragmatic surfaces and by the occasionally encountered emphysema in older laborers produced by prolonged violent inspiration without significant obstruction.

Simple description of the two factors found in pulmonary emphysema is of course not adequate to understanding of its etiology. In the first place although one factor or the other is usually predominant both are usually operative. The heavy incidence of pulmonary emphysema as compared to other equilibria severe infections such as virus pneumonia, as compared to other an organism is a co-existent factor in the emphysema in which the predominant factor is over-distention. In the same way atrophic emphysema in which the predominant factor is atrophic degeneration of the alveolar walls is commonly encountered in men whose occupations would suggest chronic

over-distention. An inherited alveolar weakness is frequently strongly suggested by familial history. In practice although either chronic over-distention or alveolar degeneration will be etiologically predominant it is impossible to set up a simple cause and-effect relationship.

A more important difficulty to etiologic relationship frequent phenomena of compensation which is concealed.

functional at 1

collections

beyond the

emphysema we must recognize even though we cannot assess the alveolar distention as a dynamic development a response at least partly compensatory in origin. In this connection it may be noted that an area of obstructive emphysema may be accompanied by compensatory emphysema.

Pathology — The situation of partial block in the alveolar walls once begun continues presence of the compress the blood vessels. More important the alveolar walls tend to obliterate the capillaries by which the blood is oxygenated. The amount of residual lung increased at the expense of tidal air. The amount of residual lung contains blood oxygen.

in press

inspiratory

inhibiting

The so-called

in elderly

hyper-expansion

and the

the

gr

an

At autopsy

much high

in efficiency

crepitant

per trachea

thickened

though few investigators have remarked upon it interstitial emphysema is probably present to quite a degree and may in part account for the failure of the lung tissue to collapse even after sectioning. The blebs characteristic of pulmonary emphysema probably represent in many instances air forced into non-alveolar tissue as evidenced by the typical location at the surface of the lung.

Where emphysema

relatively

factor is present

compress the

It is frequently noticed that the pressure in an emphysematous lung coming to autopsy is greater than atmospheric. This has in the past been

misunderstood. No conceivable check valve arrangement could produce

rotation caused by the emphysema even without an original exciting agent



FIG. 123. Last most marked in functional emphysema. The x's indicate bullae. "A" represents bullae on the under surface of the lung. "B" represents emphysema of the lung.

short interval the unaffected parts of the bronchial system force air beyond the occlusion and the emphysematous section because of lack of elasticity cannot force the air out again. Thus the pressure within the occluded emphysematous area can be maintained at higher than atmospheric pressure as long as the cough continues. This high pressure is not seen in hypertrophic emphysema where the entire lung is emphysematous.

As before remarked a high degree of emphysema may, with removal of the precipitating condition subside with no detectable clinical after-effect. This is particularly so if the cough reflex is not present or is inhibited in some way. It is probable that many instances of emphysema associated with some other infection reach the point of occlusion and then subside as the air is removed by the blood stream and penetrates the alveolar walls into unaffected parts of the lung. On the other hand the elasticity of the alveolar walls may be permanently impaired and the emphysema becomes a chronic condition terminating frequently in right sided heart failure.

Clinical Symptoms—The patient with emphysema will frequently present himself to a physician because of symptoms due to precedent or resulting infection. The chief symptom of emphysema itself is dyspnea more or less pronounced according to the area of lung involved but always out of proportion to any accompanying bronchitis. It may sometimes resemble a proximal asthmatic attack. Cough is also common but whether or not it is due to emphysema itself is difficult to ascertain.

Physical Signs—Any marked degree of emphysema will reveal itself in an enlarged chest cavity. The ribs are markedly horizontal and the sternum projects outward. Chest wall excursion is limited. If the emphysema is confined to one lung the chest wall may be obviously unsymmetrical. Severe and long standing emphysematous patients are often quite cyanotic in spite of being otherwise well nourished. The sternocleidomastoid muscles are prominent and the veins of the neck distended.

Hyperresonance is marked upon percussion. The liver will be lower than normal. In pathologic emphysema the breath sounds will be diminished sometimes being barely audible and the expiratory phase is prolonged. In compensatory emphysema there is no impediment in the air passages and breath sounds are increased. Rales and other abnormal findings are due to concurrent conditions.

Röntgen ray Findings—Two factors need to be watched for in diagnosing emphysema by roentgen ray—hyper brilliancy and indistinct markings of lung fields. Ordinarily there will be no sharp demarcations unless large blebs are viewed as air-cysts. The diaphragm will be flattened and under fluoroscopy comparatively motionless. Occasionally the thoracic cage may even be slightly concave. Distortion of the heart may be displaced.

Laboratory Findings—Laboratory techniques are not ordinarily used in diagnosis of emphysema. The only consistent finding due to the condition itself is an abnormal CO level in the blood. When a diagnosis of emphysema is established however sputum tests for bacteria should be undertaken.

Differential Diagnosis—The most important differentiations to be made in a diagnosis of pulmonary emphysema are spontaneous pneumothorax, bronchial asthma, large lung cavities, cystic or tuberculous pneumoparitoneum and diaphragmatic hernia.

Spontaneous Pneumothorax—This condition may be mistaken for unilateral emphysema. Such a diagnosis will usually be contraindicated, however, by sudden onset of symptoms, particularly dyspnea. A history of antecedent physical strain may or may not be elicited. Upon inspection, distortion of the chest wall is not usually prominent, although there may be

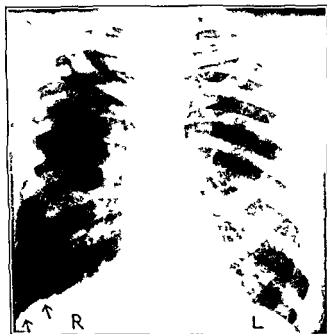


FIG. 124.—1. Seventy-one year old male, had dyspnea for many years and

had thickening of
Over the right

noticed limited area
can be elicited by
on spontaneous pu
the case in pulmon
the distinction is roentgen ray films, the separation of the lung from the

Bronchial Asthma — This condition may be suggested when a patient with hypertrophic pulmonary emphysema presents himself. Both conditions are characterized by chronic dyspnea and a protuberant chest wall. The asthmatic is much less apt to be well nourished than the emphysematous patient. Although the dyspnea in both instances is aggravated by physical or emotional disturbance, the dyspnea in the asthmatic in such circumstances is much more pronouncedly an attack. A pronounced wheeze frequently characterizes the asthmatic particularly during an attack. Asthma is accompanied by emphysema but as seen roentgenologically it is patchy and transient.

Large Cavities — Cavities may be difficult to distinguish in some in

and evidence of disease process in other parts of the lungs. Congenital cysts are also sharply outlined. Acquired air cysts are perhaps the most

92 and Fig 102 page 205)

Pneumoperitoneum — This can always be distinguished roentgenologically though physical examination may be confusing by the presence of the diaphragm high in the chest cavity, the reverse of the situation in emphysema. (See Fig 43 page 95)

Diaphragmatic Hernia — Diaphragmatic hernia can be differentiated by introducing barium into the stomach in addition to a barium enema. The roentgen ray film will definitely show that the air pocket is in the stomach or bowel. The fact that the air present is unilateral rather than bilateral also helps to distinguish this condition from emphysema. (See Fig 39 page 91)

BIBLIOGRAPHY

1. ... of emphysema by abdom
n
ty
s
th
M J 25 52 333
GOGGIO A F Abnormal physiology of chronic pulmonary emphysema 3 contrasting illustrative cases New England J Med 231 672 1944
HAMMAN L Spontaneous interstitial emphysema of lungs Tr Assoc Am Phys 52 311 1937
JAGIE N and SPENGLER G Emphysem und Emphysemherz Vmn 1924 p 2
KERR W J and LAGEN J B Postural syndrome related to obesity leading to postural emphysema and cardiorespiratory failure Ann Int Med 10 563 1936

Tamm, W. H. A. and Smith, H. L. *Am J Pathol* 1934, 21, 103-114
 Tamm, W. H. A. and Smith, H. L. *Am J Pathol* 1934, 21, 115-124
 Tamm, W. H. A. and Smith, H. L. *Am J Pathol* 1934, 21, 125-134

APRIL 3, 11, 1935, 1936

- MACKLIN, M. T., and MACKLIN, C. C. Malignant interstitial emphysema of lungs and mediastinum as an important occult complication in many respiratory diseases and other conditions: clinical literature in light of laboratory experiment, *Medicine* 23, 281, 1944
 MILLER, J. A. Pulmonary fibrosis and emphysema, *Ann Int Med* 9, 219, 1935
 MILLER, J. A. and RAPPAFORT, I. Relation of pulmonary function to fibrosis and emphysema, *Ann Int Med* 11, 1644, 1938
 NELSON, W. E. and SMITH, L. W. Generalized obstructive emphysema in infants. *J Pediat* 26, 36, 1945
 SANQUIRICO, G. Development and regression of pulmonary air cysts. *Radiol med* 32, 387, 1946

34. NON-PARASITIC CYSTS OF THE LUNG

CONSIDERABLE literature has been published on the subject of cysts of the lung since A. R. Koontz described this subject in great detail in 1925. It is now obvious that in spite of its comparatively recent description the cyst is a rather common lung anomaly.

Etiology—Most authorities now agree that it is impossible to demon-

likewise conclude that there is no reason to postulate a single origin.

The genesis of the congenital lung cysts is still debated. Sauerbruch thinks they develop as a result of a bronchus or bronchi being enveloped by pleura in prenatal life; the size of the resultant cyst would depend on the size of the bronchi involved. Some have advanced the theory that the con-

In an instance where a bronchus fails to attain complete development such as in the case of a bronchus which is only a small diverticulum. On the other hand some cysts seem to represent development of a bronchus which is completely normal. In these cases the cysts seem to occur when the bronchi develop normally but the alveolar tissue beneath does not; the resulting large air sac becomes a cyst. A similar situation but one in which the lung tissue is only a little short of complete development may possibly account for some cases of congenital bronchiectasis.

Perhaps the simplest explanation for most congenital air cysts is essentially the same as the explanation of the acquired cyst: for some reason a partial obstruction of the bronchus with valve action exists that permits the air to be inspired but not expired. The constantly mounting pressure

under of abnormal conditions
ic description of cysts is found
T. Holmes Sellers of London

Congenital Cysts

Smooth lining little contents unless infected

Regular columnar epithelium unaltered by milder degrees of infection

Acquired Dilatations

Rough dirty lining perhaps ulceration and pus formation

Columnar epithelium shows inflammatory changes with ulceration metaplasia and formation of granulation tissue

Congenital Cysts

Predilection for parts of the lung not affected by acquired lesions i. e. sub-plural areas below apices

Relationship to the line and direction of air tubes not obvious

Cysts visible in spiracular form

Small bronchi pulled out straight i. e. the lumen of the tube can be seen as a straight channel for some distance in one microscopic field

Acquired Dilatations

More centrally placed with a distribution that tends to be uniform over a lobe or lobes particularly the lower

Lie in the line of normal bronchial anatomy

Dilatation starts in the funnel form manner and tends to work down the bronchus

Small air tubes are probably tortuous and are not seen over any length in any microscopic field

Lung cysts may be solitary or multiple the former condition is most

distinction on

ive the charac-

walls may be

smooth or ribbed with septal tissue and rarely a cyst is encountered that has a wall so infiltrated as to resemble carcinoma. The pathology of cysts of the lung is obviously extremely variable

As before remarked in most cysts a connection of some sort with the bronchial tree can be demonstrated although the connecting bronchioles

are not with a certain serious nature

Thus mixed pathology seems to the author conclusive evidence of a multiple etiology

Clinical Symptoms The clinical manifestations of a condition found in such diversity are of course themselves diverse. It is possible that many cysts are asymptomatic unless complications occur. In many instances cysts are demonstrated in the course of routine chest roentgen ray examinations.

The most typical group of symptoms are found in children or infants with congenital lung cysts such cases are quite often marked by attacks of dyspnea and cyanosis caused by mediastinal shift. These symptoms may occur simply because of the large size of the cyst. Quite often a tension pneumothorax results when a cystic rupture occurs.

A cyst filled with fluid may cause mild pain, cough, dyspnea and perhaps circulatory disturbances generally of moderate nature. An extremely large cyst filled with air may manifest itself only in signs of decreased lung capacity.

In infected cysts the symptoms will depend on the type of infection and the location.

Physical Signs Physical signs in the case of small multiple cysts are oftentimes entirely lacking. Sometimes a suggestion of fibroid changes can be elicited by percussion. In the case of a large cyst of course the examiner can determine the presence of an air or liquid filled pocket within the thoracic cage.

Roentgen ray Findings — The recognition of a lung cyst that is not complicated by a secondary infection depends very largely on *roentgenoscopy*. An air cyst will appear as a clear area without the normal bronchial markings through it and it will be thinly and irregularly outlined. When small multiple cysts exist the appearance may be rather like a honey comb. Multiple cysts may be grouped around the tracheo-bronchial tree or clustered at the junction of lung and pleura rarely they are scattered irregularly.



126) Diagnosis Cysts of the lung and a right spontaneous pneumothorax



FIG. 126.—Same patient as Figure 125, after injection of iodized oil into the pleural space. Note how the cystic walls are outlined by oil, and also the edge of the lung, indicating the extent of the pneumothorax at the lung apex.

Diagnosis: Cysts of the lung with spontaneous pneumothorax (Lapwood).

roentgenogram without it perhaps merely suggested may indicate the

bronchus leading into the cyst. The fibrous bands that often transverse large cysts can be seen distinctly. However, sometimes a tracheo bronchial injection will not penetrate the cyst. Whether or not the examiner is justified in injecting oil directly into the cyst in such an instance is debatable. It does offer exact information, but some slight risk attends such an operation.

In the case of
 reveal many of
 is rotated throu

reveal many more of the bronchi and cysts, and a fairly accurate picture of the extent of the abnormality can be obtained.

Bronchoscopy—This method is not ordinarily helpful to the diagnosis of lung cysts. Sometimes constricted bronchi may indicate the nature of the abnormality and rarely the reason for the constriction will be seen.



FIG. 127. The herniation of the stomach is well outlined by barium and air.
 Diagnosis. Diaphragmatic hernia.

In some instances the bronchial tree adjoining the cyst may seem to be without bronchi.

Thoracentesis—This procedure is of value. Sometimes it is used to drain an infected or fluid-filled cavity for diagnosis; more often it is used to determine the internal pressure of cavities filled with air. Pressure at or near the atmospheric level indicates a cyst with a connection into the
 - iding that exceeds
 check valve in the

! . . . not be called upon
 if a secondary in-

fection. The examination of a specimen of the contents of a cyst of course

NON PARASITIC CYSTS OF THE LUNG

completely establishes a diagnosis but the thin walls of this growth and the danger of spontaneous pneumothorax ordinarily makes the taking of such a tissue section inadvisable.

Differential Diagnosis—In the differential diagnosis of congenital and acquired cysts of the lung the examiner must consider the possibility of diaphragmatic hernia, tuberculous cavities of the lung, emphysematous blebs, spontaneous pneumothorax, echinococcus cysts, abscesses of the lung and bronchiectatic cavities.

Diaphragmatic Hernias—A distinction between this condition and lung cysts can usually be made with some certainty by roentgenographic study. However, positive distinction can be made by introducing barium into the stomach and also into the large bowel. If a diaphragmatic hernia is present the barium will outline the stomach and the large bowel above the diaphragm.

Tuberculous Cavities—A tuberculous cavity presents a typical appearance on a roentgen ray film. Its outline will be irregular and its walls much thicker than the walls of a cyst. In addition tuberculous lesions can almost invariably be seen in other parts of the lung. Distinction can be made further on the basis of the patient's history and clinical symptoms. The presence of tubercle bacilli in the sputum is a decisive finding but is not at all conclusive since tuberculous infection may be coincidental with the lung cyst. (See Fig. 40, page 92.)

Emphysematous Blebs—Both the bleb and the bulla are formed by the rupture of pulmonary alveoli; the former however results when the tissue lies close to the outer edge of the lung and so ruptures in the pleura. It may easily be mistaken for a bulla since a bulla will distort the intervening alveoli in many cases and project into the pleura. (See Fig. 128, drawing.)

Method of differentiating the two conditions is by creating an artificial pneumothorax so that the air space of the sac the oval shape of the bulla is seen. This procedure is seldom needed in the roentgenogram. (See Fig. 41, page 93.)

Spontaneous Pneumothorax—A pneumothorax often results from the rupture of a cyst and the conditions are sometimes hard to differentiate. Repeated attacks of cyanosis and respiratory distress usually indicate a pneumothorax. In a roentgenogram the adhesions of a pneumothorax are curved and a cyst are curved. An artificial pneumothorax is usually of a

id has ruptured it may look much like a non parasitic cyst partially filled with fluid. Differentiation can be made on the basis of sputum tests. A ruptured hydatid cyst will almost always secrete into the tracheo bronchial tree a thin clear fluid containing the characteristic scolices by which it reproduces itself. (See Fig 82 page 165.)

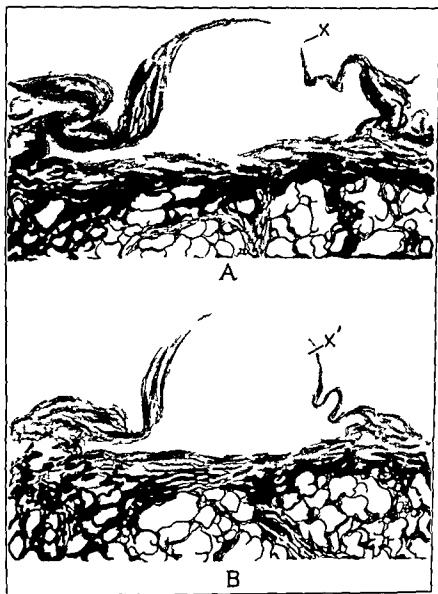


FIG 128 —A schematic drawing showing enlarged vesicles in the lung representing varying sizes of bullae. In A area X represents an unruptured bleb. In B the bleb has ruptured producing a spontaneous pneumothorax.

Abscess of the Lung—The severe clinical symptoms and the history of previous pulmonary infection will serve to distinguish this condition from the cyst. If the cyst is infected, however, it may be impossible to differentiate these two conditions. In this case it makes little difference since prognosis and treatment are the same for the two conditions. (See Fig 102, page 205.)

Bronchiectatic Cavities—If the cyst is infected, it will likewise be difficult to distinguish from bronchiectasis. The bronchiectatic condition can usually be determined by roentgenography following the injection of iodized oil, and again the question of origin is unimportant to treatment.

A further possibility of confusion exists in the similar congenital origin of a form of bronchiectasis and of a form of congenital cysts. (See Fig 74, page 156.)

BIBLIOGRAPHY

- ADAMS, W. F., and THORNTON, T. F., JR. Bronchogenic cysts of mediastinum with report of 3 cases, *J. Thoracic Surg.* 12, 503, 1943.
- BALLON, H. C., SINCER, J. J., and GRAHAM, F. A. Bronchiectasis, *J. Thoracic Surg.* 1, 151, 1931.
- CORYLLOU, P. N., and BIRNBAUM, G. I. Studies in pulmonary gas absorption in bronchial obstruction, II, *Am. J. M. Sc.* 193, 329, 1932.
- EISENBERG, I. Congenital cystic disease of the lung. *S. Clin. North America* 8, 1361, 1928.
- GIBBS, J. S. A. C.
- GRAHAM, F. A.
- KOO,
- LEVIN,
- OLIVER,
- 1936
- SAMERBRUCH, F. *Die Chirurgie der Brustorgane*. Berlin, Julius Springer, 1925.
- SELIERS, T. H. Congenital cystic disease of lung. *Tubercle* 20, 4) and 114, 1938.
- SINCER, J. J. Tumors and cysts of the lung, diagnostic methods. *Calif. & West. Med.* 45, 313, 1936.
- Acquired cysts of the lung, clinical and X-ray study. *Bull. Am. Acad. Tuberc. Physicians* 5, 2, 1911.

id has ruptured it may look much like a non-parasitic cyst with fluid in it.

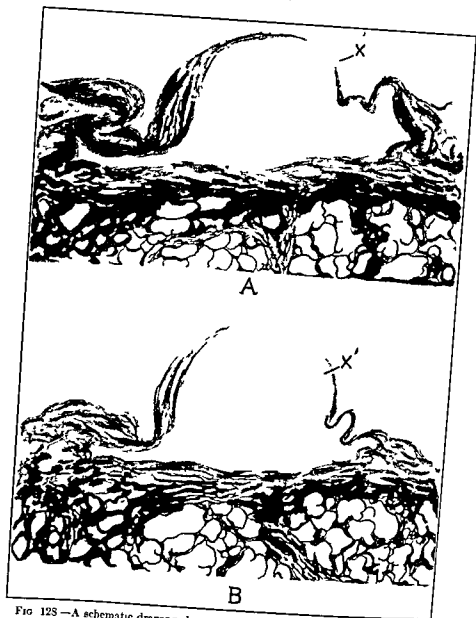


FIG 128 — A schematic drawing showing enlarged vesicles in the lung representing varying sizes of bullae. In A area X represents an unruptured bleb. In B the bleb has ruptured producing a spontaneous pneumothorax.

Abscess of the Lung —The severe clinical symptoms and the history of previous pulmonary infection will serve to distinguish this condition from the cyst. If the cyst is infected however it may be impossible to differentiate these two conditions. In this case it makes little difference since prognosis and treatment are the same for the two conditions. (See Fig 102 page 205.)

Bronchiectatic Cavities —If the cyst is infected it will likewise be difficult to distinguish from bronchiectasis. The bronchiectatic condition can usually be determined by roentgenography following the injection of iodized oil and again the question of origin is unimportant to treatment.

A further possibility of confusion exists in the similar congenital origin of a form of bronchiectasis and of a form of congenital cysts. (See Fig 74, page 156.)

BIBLIOGRAPHY

- ADAMS W E and THORNTON T F JR. Bronchogenic cysts of mediastinum with report of 3 cases. *J Thoracic Surg* 12 503 1943.
- BALLON H C, SINGER J J and GRAHAM F A. Bronchiectasis. *J Thoracic Surg* 1 154 1931.
- CORYLLO P N and BIENBAUM G L. Studies in pulmonary gas absorption in bronchial obstruction II. *Am J M Sc* 193 326 1932.
- FLOESSER I. Congenital cystic disease of the lung. *S Clin North America* 8 1361 1928.
- SAUERBRUCH F. Die Chirurgie der Bronchorgane. Berlin Julius Springer 1925.
- SELLORS T H. Congenital cystic disease of lung. *Tubercle* 20 49 and 114 1939.
- SINGER J J. Tumors and cysts of the lung. diagnostic methods. *Calif & West Med* 45 313 1936.
- Acquired cysts of the lung. clinical and X ray study. *Bull Am Acad Tuberc Physic ins* 5 2 1941.

35. THE HYDATID CYST OF THE LUNG

THE hydatid or echinococcus cyst is relatively rare in North America although each year a few patients afflicted with the parasite present themselves to the larger clinics. In all but a few instances in the past these patients have been immigrants from the countries where the disease is common—Italy, Australia or Argentina. A larger incidence among the native population can be expected in the next few years however due to the number of armed services personnel stationed in these countries during World War II.

In spite of its infrequent occurrence the possibility of a hydatid cyst should be kept in mind in making a diagnosis. It is readily confused with other lung diseases and its nature is such that treatment under mistaken

*in this country because of the un-
familiarity with echinococcus.* The fulfillment of this cycle requires not only a stockraising situation but one in which sanitary conditions with regard to butchering are much more lax than ordinarily prevails in this country. The cycle starts with the elimination of the ovum in the feces of a dog, jackal or wolf. These ova must then be ingested by sheep or less commonly cattle or pigs. The ovum then matures in the viscera of the sheep and the cycle is completed when the dog, wolf or jackal eats the infected viscera so that the parasite is again lodged in canine intestines.

Man becomes infected by the ovum from the dog's feces. It is now generally thought that this infection must be through the alimentary system. The acids present in the stomach dissolve the envelope in which the echinococcus larva is enclosed and it starts to grow. Probably the echinococcus remains in the alimentary tract until it gets to the liver because that is the site of an overwhelming percentage of the cysts found

ent location
to indicate
1 1 1

ism begins to feed on surrounding tissue and grows rapidly. Usually there is only a single cyst although two or more may be found. Very rarely small cysts of a uniform size will be found scattered rather evenly around the periphery of the lung. Multiple grouped cysts are usually found. Daughter cysts occur to the hydatid body but no true daughter cyst has

h a thin clear alkaline fluid with a
15 It contains sodium chloride glu

by which the cyst is reproduced

This fluid is enclosed by a germinal layer of tissue which forms the brood capsules the scolices and daughter cysts and is apparently the ingesting mechanism for the organism. It is quite thin. In a cyst of any size this laminated membrane is in turn enclosed by a tough white membrane which accounts for many of the characteristics of a hydatid cyst because of its resistance to infection and drugs.

The final phase of cystic development in man may take any one of several directions. Rarely the cyst will grow so slowly as to allow the adventitious tissue around it to calcify and thus kill the cyst. The cyst degenerates

newly in this case the adventitious tissue becomes fibrotic and shrinks into a lesion much like that of tuberculosis. The most common end, however

become quite large and remain asymptomatic. The first appearance of symptoms often follows the subsidence of another acute respiratory infection. Cough and a slight hemoptysis are usually the earliest indications of the abnormality. Pain is not uncommon if the pleura is involved and it may be referred to other parts of the viscerum. A large cyst by mere size may produce dyspnea.

The cyst frequently first comes to the attention of the physician when it ruptures. If the cyst is small and located near a large bronchus the fluid and membrane may both be coughed up and a spontaneous cure may result. This occurs in a surprising number of instances. In a majority of cases however more or less serious symptoms attend the rupture of the cyst. Anaphylaxis characterized by cardiac disturbances, paroxysmal dyspnea, pruritis, cyanosis, etc. is very frequent and in some cases it is fatal. The degree of severity of the anaphylaxis bears no relation to the amount of

usually chest dullness to flatness depending on the relation of the cyst to the chest wall and indicating certainly a large amount of fluid in the under-

THE HYDATID CYST OF THE LUNG

of other visceral organs will be found by physical signs and some restriction of motion of the affected side may be noted

Roentgen ray Findings — A simple hydatid cyst of the lung will appear on the roentgenogram as a homogeneous shadow completely round except for the indentation of visceral organs in the area. For the most part the shadow will be quite sharply defined although there may be patches of fuzziness around it due to local inflammations of the adventitious tissue. Sometimes the Escudero Neimeno sign in which the shape of the shadow changes slightly during respiration is seen. If serial roentgenograms are taken the cyst may be seen to grow rather rapidly.



FIG 129 — A. A four year old white female
The patient entered hospital with a history of a bad cold and a ray showing
growth in the lungs one year prior to entry. She was born and raised near Reno, Nevada.
Tuberculin 1:1000 negative
Echinococcus skin test negative
Thoracotomy revealed an echinococcus cyst
Chest roentgenogram reveals a large area of radiolucency enclosing a fluid level in
the left lower lung field. Below this there apparently is fluid in the left costophrenic
angle.
Diagnosis Echinococcus cyst of the lung

When a bronchus has been eroded as mentioned earlier, certain very typical roentgenographic appearances are noted. The first development is a thin layer of air visible around the top and sides of the cyst. This layer of air is due to the escape of gas from the cyst into the surrounding tissue.

degeneration of the cystic wall continues until it collapses. A roentgenogram of the thorax which is taken at this stage shows a typical appearance.

Unless a spontaneous cure is effected or unless there is surgical intervention an hydatid cyst which is ruptured almost inevitably becomes infected and develops the typical roentgen ray appearances of a chronic pulmonary abscess. Rarely multiple secondary cysts result from such rupture at the site of the original cyst. In a few cases rupture of a lung cyst may result in secondary metastatic cysts in other parts of the body which may be discovered roentgenographically.

The rare condition of calcification of adventitious tissue around a slowly growing cyst is readily identifiable by roentgenological means since this is one of the few instances of lung calcification encountered and the thick circle is unmistakable. Patches of ossification may also be seen in such a circle occasionally.

The examiner may have some difficulty in locating the cyst as to its presence in the lung, pleura or diaphragm. If it is possible such a distinction should be made on the basis of physical signs and roentgenograms. Lateral projections will be of particular help. Because an hydatid cyst is easily ruptured and because rupture may be attended with such

eosinophil count is not present in the blood when the cyst is dead or infected but it is very often found so long as the cyst is alive.

The Casou intradermal test is the most frequently used laboratory test when hydatid disease is suspected. In this test a small amount of fluid from hydatid cysts in sheep is injected into the forearm of the patient. A positive reaction consists of a raised wheal with outrunner reaching its full development in about twenty minutes with a secondary reaction some hours later of eosinophilia in the area with sub-adjacent induration. Barrett and Thomas say that a positive reaction indicates a cyst about

A skin test gives definite conclusions can be drawn

is found mixed with sputum it will very often be infected and its distinguishing characteristics obscured. Direct aspiration of the fluid should never be attempted since the anaphylaxis earlier described often follows such aspiration.

Bronchoscopy—A bronchoscopic examination will ordinarily be of little help in diagnosing an hydatid cyst. If the cyst is large evidence of the displacement of the tracheo-bronchial tree and other organs may be seen and if it is ruptured the ulceration in the bronchi may be noted.

Differential Diagnosis—Perhaps because it occurs so rarely in this country that physicians fail to consider it this cyst has been diagnosed at one time or another as almost every other lung abnormality known. Barrett and Thomas even report two cases in which the rash of echinococcus anaphylaxis was mistaken for measles rash. The hydatid cyst of the lung and pleura is most frequently mistaken for a carcinoma or benign tumor of the lung, an encapsulated empyema, aortic aneurysm, atypical pneumonia, pulmonary tuberculosis, non-parasitic cyst, tuberculous pleurisy with effusion and a ruptured hydatid cyst of the liver.

The geographical history of the patient is most important to differential diagnosis. The great majority of patients presenting themselves to physicians with unidentified lung abnormalities will not have been exposed to the stockraising situation with persistently inadequate sanitary conditions that this parasite requires. However when a patient has been for some time in countries where the disease flourishes the possibility of hydatid cyst needs to be considered.

The second general consideration to differential diagnosis is the laboratory findings. When a Casoni test is negative the physician can except in the case of very strong contraindications eliminate the possibility of an hydatid cyst. On the other hand a positive Casoni with a positive Ghidini Wernberg and eosinophilia establishes the disease with a considerable degree of certainty.

Thoracic Tumors—An uncomplicated cyst can be readily mistaken for

is especially likely to be more severe. If the lesion is large enough physical signs significant the difference between a solid tissue and a liquid filled tissue may be noted. The effect of roentgen ray treatment on the abnormality may be determined; no reaction is ever obtained from a cyst. If it is a malignant tumor fluid aspirated bronchoscopically may be found to contain tumor cells.

is a homogeneous
fluid
type

round shape of a cyst (See fig 166 page 312.)

Encapsulated Empyema—Distinction between an encapsulated empyema and a cyst is suggested by the patient's history. When there is a history

of previous pneumonia or other suppurative conditions of the lung the symptoms are more apt to be due to an encapsulated empyema. If the patient has not been exposed to a situation in which the echinococcus might flourish the physician can perform with confidence a diagnostic

10-
an
he
ig

(4 page 121)

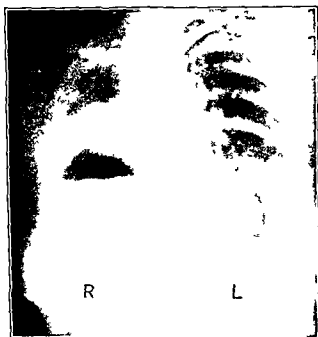


FIG. 130 - *Diagnostic pneumothorax* Film taken after the aspiration of 400 cc. of fluid by the diagnostic tap at a distinct

Differentiation can also be made on the basis of physical signs since it is almost always possible to detect the characteristic sounds of an aneurysm by auscultation. Further evidence will be found in an analysis of the blood usually eosinophilia is not present in aneurysm syphilis is in a great majority of cases.

Atypical Pneumonia - This may be confused with a ruptured and infected cyst where clinical symptoms of low grade fever and so forth are present. When either of the two conditions may exist by other evidence,

sputum examination for scolices and cystic debris is indicated and will confirm a diagnosis if the cyst has ruptured into the bronchial tree (See Fig. 97, page 194.)

Pulmonary Tuberculosis — As before remarked when a cyst dies and degenerates without establishing a connection with the bronchial tree the resultant lesion looks roentgenoscopically, very much like a tuberculous lesion. Such a lesion will be single however and a tuberculous lesion of



such a size would almost never occur without evidence of tuberculosis in the surrounding tissue. A bronchoscope may confirm the fact that the lesion has no opening into the tracheo-bronchial tree and secretions from the area will be free of tubercle bacilli. The Casoni test will be of no value in this differentiation since active tuberculosis frequently also gives a positive finding.

Non parasitic Cyst — This would be most readily mistaken for a degenerated cyst where the cavity has maintained the typical circular shape. In some cases the degenerating cystic wall of the hydatid can be seen in the roentgenogram or the sputum may contain the echinococcus organism. Further non parasitic cysts seldom show symptoms unless the cyst is

quite large or infected (or unless spontaneous pneumothorax develop
(See Fig 38 page 90)

Tuberculous Pleurisy — This disease may resemble a ruptured echino-

Ruptured Hydatid Cyst of the Liver—A cyst of the liver is most frequently located in the upper right lobe of that organ. Not uncommonly such a cyst will push upward displacing the diaphragm which becomes flat and immobile. In some cases an hepatic cyst will actually encroach upon the

in the liver by bile in the sputum

BIBLIOGRAPHY

- [illegible]

36. PULMONARY TUBERCULOSIS

MEDICAL ASPECTS

TUBERCULOSIS is the most common disease of the lungs and it has received the most attention. In spite of the voluminous research that has been done and in spite of the publicity that has been given this specific lung infection the typical industrial mass roentgenologic study reveals from $\frac{1}{2}$ to $1\frac{1}{2}$ per cent of the group surveyed has pulmonary tuberculosis not previously diagnosed.

With the discovery of antibiotics within recent years we have finally at hand a weapon with which to fight the disease directly. It is almost certain that the next few years will see the known drugs available to all tuberculous patients and it is possible that even more effective remedies will be developed. We are now in a position for a final assault.

The major part of the disease is caused by a non-specific general practitioner because of its being difficult to combat by a large measure still the general practitioner.

The standard system of classification of adult type tuberculosis is based upon roentgenologic findings. A minimal lesion is one without demonstrable excavation and with total involvement not exceeding the equivalent lung volume of the tissue above the second chondrosternal junction and the spine of the fourth or body of the fifth thoracic vertebra on one side. A moderately advanced lesion is one which does not present severe involvement of more than one-third of one lung or infiltrative or fibrotic involvement to exceed the volume of one entire lung. A far advanced lesion is one exceeding a moderately advanced lesion or a lesion presenting definite cavity formation. This system does not take into consideration clinical symptoms which oftentimes do not correlate with roentgenologic findings.

Etiology—In the enormous amount of research done since the isolation of acid fast bacilli several distinct strains have been isolated. Saprophytic forms are found throughout nature but are so far as is known completely

ogenic for most mammals. In man the lesions of this bacillus are usually

where milk is not pasteurized. At least 90 per cent of all cases of human tuberculosis in the United States are due to the human type bacillus. The bacilli can infect any organ of the body, but the initial infection is pulmonary in almost all instances.

Distinction between these varieties of *Mycobacterium* are made largely on the basis of pathogenicity for laboratory animals. By complicated laboratory techniques further strains within these primary groups can be differentiated. The author will discuss unless otherwise indicated the

into extremely fine particles from which new rods appear to sprout.

The mechanism of infection is still a matter of some debate. It seems likely that the bacilli normally gain entrance to the lungs by means of contaminated air borne dusts. The amount of infection contracted by direct inhalation of droplets of contaminated sputum is probably small since it has been demonstrated that droplets of sputum small enough to remain suspended in the air and small enough to evade the protective mechanisms of the respiratory tract ordinarily contain few or no bacilli.

Few persons have a natural resistance to the invasion of tubercle bacilli. This is indicated by the extraordinarily high percentage of primary infections in individuals from a crowded tuberculous environment. The individual response to this initial lesion is however the crucial factor. In a great majority of persons a localizing immunizing reaction together with an allergic response develops and the infection is brought under

for
nity
de
in

fection.

When a primitive group is first exposed to tuberculosis immunity does not develop and the initial lesion progresses to massive overwhelming

that a substantial percentage of the population at one time or another become infected with acid fast bacilli and in some urban areas the percentage of non reactors has been so small as to be insignificant. Quite com-

usually sub-pleural. Caseation demonstrated roentgenologically is fairly rapid but remains rather circumscribed. Typically a cellular exudate is found in nearby alveoli and in many instances a rather widespread edema develops in the area of the lesion. Involvement of the lymph nodes draining the area is almost invariable and follows shortly after the parenchymal

PULMONARY TUBERCULOSIS—HISTOLOGICAL ASPECTS

infection develops. Such lymph nodes often become greatly enlarged and caseous with bacilli present in great numbers. This combination of primary chymal lesion and enlarged lymph nodes is the Ghon complex. Lesser involvement in contiguous lymph systems is very often demonstrable by microscopic examination.

It is now apparent that in most instances of primary infection some degree of hematogenous dissemination does occur. Small tuberculous foci are frequently demonstrable in other organs in instances of healed primary complexes. The status of the commonly found apical scars which were formerly attributed to post primary hematogenous spread is however in some doubt. Medlar's study suggests that at least in many instances such scars are of a non tuberculous origin.

The development of immunity as remarked under Histology is still a mysterious process. Occasionally such a reaction is absent even in individuals of groups long exposed to tuberculosis. In such instances the hematogenous spread may involve almost every organ of the body with tuberculous meningitis the usual cause of death.

The typical primary complex is richly encapsulated by connective tissue fibers. The pulmonary lesion may be absorbed but fibrosis is the more oftentimes incomplete indication of both lesions. The lymph nodes are particularly apt to prevent evidence of incomplete reparative processes with potential exacerbation many years later.

This local mobilization against infection probably depends upon a systemic mobilization for its effectiveness. After the development of systemic immunity no further spread of the bacilli by lymph or blood occurs. This early phase of primary infection is seldom ever seen clinically seldom severe enough or prolonged enough to come to the attention of a physician.

In most instances a primary infection produces a lasting immunity of some degree of effectiveness. Occasionally one sees at autopsy however two primary complexes of different age indicating that the immunizing effect of the first had entirely disappeared before reinfection. Such a finding is comparatively rare.

Reinfection or adult type tuberculosis is pathologically quite different from a primary complex. The systemic defenses remain effective through limited to the organ of first infection and contiguous and contiguous connected structures without lymph or hematogenous spread. On the other hand the local defense mechanisms are usually less effective permitting progressive destruction of the organ.

The relation of immunity to reinfection is still not clearly understood. It is of course well known that physiologic and pathologic disturbances such as pregnancy, malnutrition or nervous strain may depress the immunity reaction and are thus a factor in reinfection. On the other hand it is thought that comparatively large doses of acid fast bacillus are necessary for infection even when immunity is lessened. Few deny that reinfection may be exogenous, the result of the inhalation of more bacilli.

in instances where the individual is exposed to an extremely unhygienic and tuberculous environment. However many investigators doubt that the majority of reinfection tuberculosis cases are exposed to a sufficient number of acid fast bacilli in a sufficiently short time for them to overcome the induced immunity. Such students suggest that much reinfection tuberculosis is endogenous. They point to the length of time that acid fast bacilli can live in a state of inactivity within the body and particularly in the lymph system and suggest that massive doses of bacilli such as are predicated as necessary to reinfection are most likely to result from reactivation of partially calcified primary lesions or from reactivation of post primary hematogenous foci. The typically apical sub apical or infracapillary location of tuberculosis lesions seems to indicate an endogenous and probably hematogenous origin of the disease although these locations have been used as evidence for exogenous reinfection.

In either case endogenous or exogenous an early reaction of the lung to acid fast bacilli is a cellular exudate. Animal experiments indicate that a

origin in the phagocytes according to Rich they represent a fusion of such cells. The microscopic appearance of the tubercle is due to the cluster

in edematous reaction which may sometimes be extensive.

The progress of the small tubercle from this point is variable. Pinner points out that we may presume such lesions to be arrested and resorbed at times. The number of bacilli at the center of the lesion is probably an

the

it

until

more or less constant. Once a focus of such material is established in the lung the disease process becomes a contest between the isolating and reparative powers of the body—encapsulation, fibrosis and calcification and the destructive powers of the bacilli. In many lesions all processes may be operating simultaneously.

The escape of the caseous liquid is the beginning of the tuberculous cavity which may gradually enlarge until it includes the whole lung. Spread is by contiguity and by the movement of the contaminated caseous material through the bronchi. All tissues connected by open passages to

the infected area—the bronchi the larynx and so forth—may be infected but in most cases there is no spread by means of the lymphatics or by the blood stream even where hemoptysis indicates erosion of blood vessels by the necrotic process

Clinical Symptoms The onset of tuberculosis is usually insidious. In the mass roentgen ray surveys of recent years from one-quarter to one-half of the persons found to have tuberculosis reported no clear-cut symptoms. Lassitude and a slight weight loss were the characteristic symptoms of many of the remainder.

When there are symptoms they may be of three general kinds. Those due to toxemia are loss of strength and endurance, lack of appetite or nervous instability and rapid pulse. The tuberculosis itself may produce chest pain. Sometimes symptoms of reflex origin are encountered such as hoarseness, cough or laryngeal disturbance.

It is often difficult to date the onset of symptoms. Transitory chest pain is frequently the first symptom to alarm the patient. Cough is often

discovered accidentally. It may come to a physician's attention because of a sudden chest pain of some severity, more often the patient seeks medical aid.

Cough is from lobar pneumonia prominent but non-productive in most instances. Dyspnea is not as prominent as in lobar pneumonia.

The distinguishing features of hematogenous tuberculosis are those of toxemia.

Physical Signs—Functional disparity between the two sides of a pa-

isthmus formerly greatly emphasized is so difficult to detect that it has been largely abandoned.

The protean character of tuberculosis makes auscultative and percussive findings difficult to evaluate in diagnosis. Widespread hematogenous or especially miliary tuberculosis may manifest no physical findings at all. Frequently a heavily fibrosed lung will emit nearly normal lung tones due to the effect of a compensatory emphysema. The most useful signs in early tuberculosis are diminished breath sounds with some prolongation of

resonance a so-called cavernous sound elicited by percussion and auscultation. There may be some mediastinal and cardiac displacement toward the affected side. Impaired motion with pleural rub may be prominent. An active exudate process is indicated by large areas of dullness. In tuberculous pneumonia the area of dullness may extend over a whole lobe or even an entire lung.

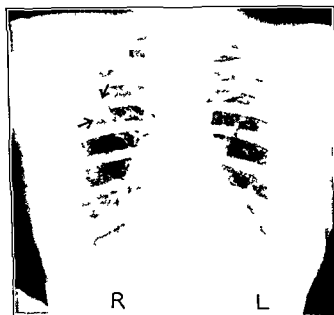


Fig. 132—Oct. 11, 1928, 10 a.m.

Fig. 133
Diagnosis: Incipient pulmonary tuberculosis

Roentgen ray Findings—Because symptoms are characteristically absent or sub-clinical in patients with a primary tuberculous infection the development of such infection is seldom followed roentgenologically. The information that we have is for the most part derived from routine studies in children's institutions.

It is thought that roentgenologic evidence of first infection appears from two to three months following exposure. The shadow when first seen is that of an exudative process; it is homogeneous but irregular in outline.

and resembles a pneumonic shadow. It may for a time occupy a whole lobe due to edema or atelectasis but clearing is rapid. After the generalized process has cleared the residual shadow is rather small.

FIG. 15. 5511

R

I



The usual end of the primary complex is calcification to a greater or lesser degree. Calcification usually proceeds more rapidly in the lymph nodes but such calcification is more likely to be incomplete and to permit exacerbation later. In some instances the parenchymal lesion may be resorbed to such an extent as to be roentgenologically invisible although enlarged and calcified hilar lymph nodes remain to suggest primary infection. Very infrequently patients have come to autopsy with demonstrable primary tuberculous lesions in the parenchyma but without macroscopic evidence of lymph node involvement. There is thought to be a rather long

(two to three months) time interval between infection and roentgenologic evidence in reinfection tuberculosis also. Unlike first infection which may appear in any part of the lung reinfection tuberculosis has a decided predilection for the sub apical or infraclavicular area. The initial shadow is usually irregularly round or fan shaped with indefinite outlines. Except for its location it is not suggestive of any particular process. The most characteristic and suggestive finding is a faint shadow from the lesion to the bronchus the drainage band. A bilateral process is not an unusual

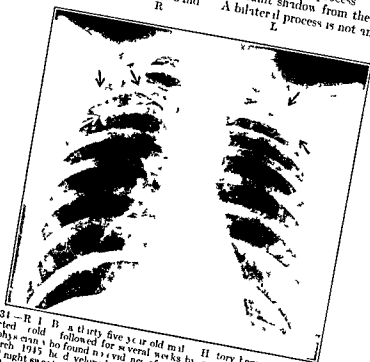


FIG. 131.—R. B. a thirty five year old male. History began in July 1944 with a protracted cold followed for several weeks by cough. He was examined at the time by physicians who found no evidence of pulmonary disease. On March 1945 he developed a cold which was followed by severe productive cough and night sweats. He entered a sanatorium 6-5-45. Physical examination revealed diminished resonance over both upper lobes with rales more marked on left side and bronchial breath sounds in left apex. Sputum was positive for acid fast bacilli. This roentgenogram 6-12-45 shows both apices to be cloudy and a cavity 1 inch in diameter in left apex. Diagnosis Bilateral pulmonary tuberculosis.

finding even in the early stages of infection but development is ordinarily unequal between the two sides. Evacuation is suggested by hazy irregular outlines when the process is predominantly fibrotic the shadows are more even and distinct in outline. The adjacent lymph nodes ordinarily show no microscopic evidence of involvement. The original lesion enlarges rather slowly spread is intracavicular for the most part. Moderately advanced tuberculosis is characterized by numerous but small shadows contiguous oftentimes but distinct. The

original lesion can usually be identified by a greater degree of fibrosis. It is not unusual to find in addition the lower lobe of the lung opposite the original site also infected. Where the infection is low grade the shadows may be predominantly of linear fibrosis marked by small discrete nodulization.

Cavitation is a prominent feature in most instances of advanced tuberculosis. A new cavity or one in which fibrosis is not an effective process

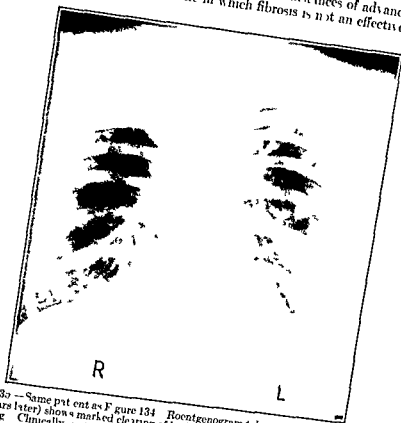


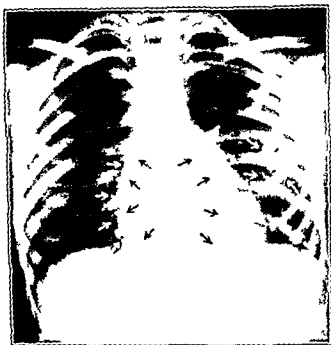
FIG. 137—Same patient as Figure 134. Roentgenogram taken 4-23-48 (approximately three years later) shows marked clearing of both upper lobes. No evidence of cavitation remaining. Clinically patient apparently arrested.
Diagnosis: Apparently arrested pulmonary tuberculosis.

is seen as an irregularly shaped area of rarefaction. Where the cavity is to some degree encapsulated the outlines will be more regular and the walls will be thicker. A fluid level may or may not be present depending upon the establishment of a communication to the bronchus.

When the mediastinum is mobile it is frequently displaced toward the unaffected side by spontaneous pneumothorax or to the affected side by the collapse of a large cavity or by fibrosis. The diaphragm, however, is obliterated on the side of the disease. Pleural adhesions obliterate the space between the pleurae. Where the phrenic nerve is involved by fibrosis or enlarged lymph nodes the diaphragm may

be seen to be high in the chest cavity (See Fig 100 page 209 and Fig 112 page 221)

ALL + m + not re-act



For a positive reaction, an area of induration or edema of 5 mm or more in diameter is considered a positive reaction. Patients who are nonreactors

to the first dilution are retested at forty-eight hour intervals with tuberculin 10 times as concentrated and with successively stronger dilutions until the final dilution 1:10 OT (10 mg) is reached. Testing with the successively more concentrated tuberculin makes it possible to assign each patient to a 'sensitivity level' or sensitivity group depending upon the dilution of tuberculin to which he reacts.



FIG. 137 Same patient as in Figure 136. Note destruction of the vertebral bodies at left arrows. Abscess cavity at larger arrows.
Diagnosis: Pott's disease and pulmonary tuberculosis.

The new preparation of tuberculin P.P.D. (Purified Protein Derivative) is a new product which is used more for testing than for treatment. Each tablet may be dissolved in 1 cc. of buffered sterile saline solution and 0.1 or 0.2 cc. of this solution is used. It should be used intracutaneously. It is much more stable and represents the tuberculin in its purified form. The method of use is: dilution of the stock solution so that 0.1 cc. will represent 1:100,000 of a mg. By mathematical computation one can easily make any dilution necessary for injection. It is advisable to have the amount of tuberculin desired in 0.1 and not more than 0.2 cc. solution so that the

mechanical effect of the quantity of fluid under the skin does not give a false reaction

In the present downward tendency of the level of tuberculization in our

cult test does not definitely exclude the tuberculous origin of demonstrated pulmonary lesions which although they are presumably perfectly healed



may yet be rekindled upon undue exposure. As the criterion of tuberculous disease we must still consider the clinical phenomena of disease

of tuberculosis and must be ruled out before a diagnosis can be made
 the
 in all
 in tum

or gastric washings must be made. The examiner should not forget the possibility of the coexistence of tuberculosis with other pathologic conditions.

Bronchiectasis—Tuberculosis ordinarily presents more persistent symptoms of debilitation—fever, chills and sweats—than does bronchiectasis in which such symptoms are ordinarily irregularly intermittent. Dry bronchiectasis with hemorrhage may otherwise be difficult to distinguish



FIG. 139.—Lateral view of the same patient as in Figure 138. Note the destruction of vertebral bodies.

from tuberculosis. Location of the lesion in the lower lobes will be suggested by history, roentgen ray appearance, and bronchoscopic examination will be suggested by history (page 157). Primary tuberculosis with which silicosis can be confused in almost all instances presents very acute

symptoms. The roentgen ray shadows of silicosis are, except where secondary infection is prominent, much denser and more sharply outlined than those of military tuberculosis. Instances in which silicosis has occurred without a readily elicitable history of exposure to silicious dusts are very rare. The coexistence of tuberculosis and silicosis, however, is extremely common. The roentgen ray appearance is largely that of silicosis the tuberculosis must be established by demonstration of acid fast bacilli (See Fig. 83 page 166.)

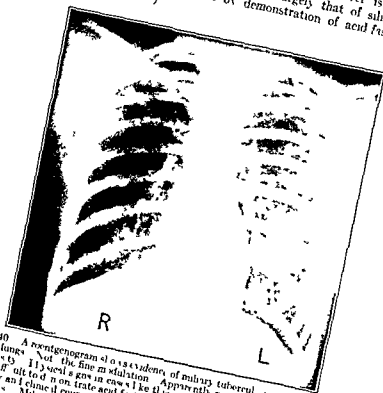


FIG. 140. A roentgenogram showing evidence of military tuberculosis scattered throughout both lungs. Not the fine mottled appearance. Apparently most of the nodules are of equal density. The silicosis in cases like this are usually negative, and it is very frequently difficult to distinguish acute and chronic bacilli. Hence one must at times depend upon the history and clinical course for definite diagnosis.

Diagnosis. Military tuberculosis (pulmonary).

Mycoses. Although the mycoses have a predilection for hilar and basal location as opposed to the typical location of tuberculosis in the upper third of the lungs, neither tendency is strong enough to be diagnostically useful. Mycoses will be suggested by pronounced enlargement of the lymph nodes and hilar thickening. None of the mycoses present the extensive fibrosis that is often found in tuberculosis. In differential diagnosis depends upon the demonstration of one organism or another in sputum. Whenever there is consistent failure to demonstrate acid fast bacilli in the sputum of patients presenting the symptoms of tuberculosis, a search for fungus should be undertaken (See Fig. 117 page 232.)

Lung Abscess—It may at times be very difficult to eliminate this possibility than
Fibrosis
made by the demonstration of the pathogenic organisms (See Fig 60, page 117)

Tumors of the Lung—Early carcinoma may be highly suggestive roentgenologically of tuberculosis and so the possibility of malignancy must



FIG 141 R N age seventy three white male rectal

The patient entered hospital with a history of bloody diarrhea for three months a thirty pound weight loss in two months a productive cough for one month

Proctoscopy and biopsy reveal adenocarcinoma of the rectum

Autopsy revealed adenocarcinoma of the rectum with widespread metastases including metastases to the lung

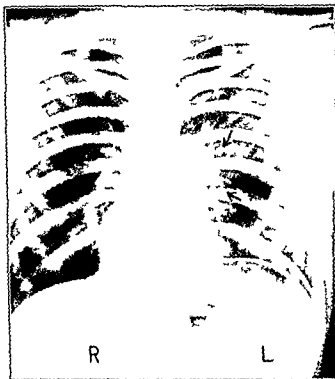
This chest roentgenogram reveals rounded nodular densities throughout both lung fields The haziness at the left base is suggestive of inflammatory disease (Note arrows)

Diagnosis Adenocarcinoma of the rectum with pulmonary metastases

be at all times the primary consideration until it has been definitely eliminated as a possibility This is particularly the case if the patient has reached middle age Diagnosis should not depend upon prolonged attempts to demonstrate acid fast bacilli in the sputum The great majority of lung cancers are hilar and only infrequently are they unapproachable by bronchoscopic means The tissue specimens thus secured will be in almost all

instances decisive. Studies of sputum for evidence of malignancy cells will be useful in differentiating tuberculosis.

The Pneumonia. Acute tuberculosis may be mistaken for lobar pneumonia. However, the area involved is usually much greater in tuberculosis, the fever is much more irregular, and the sputum is usually frankly hemorrhagic rather than rusty. Routine blood tests will be significant.



with several

the pronounced leukocytosis of lobar pneumonia is not found in uncomplicated tuberculosis. (See Fig. 2, page 71.)

Bronchopneumonia is more apt to suggest itself in instances of tuberculosis than lobar pneumonia. Friedländer's pneumonia, particularly, is

Singer, J J

Arch Phys Therap 12, 526, 1931

SING

120 1936

Roentgenological aspects of non tuberculous pulmonary disease, Am Rev Tuberc 38, 680, 1938

SOBER, W B, and AMBERSON, J B, Jr Pulmonary tuberculosis in young adults particularly among medical students and nurses, Am Rev Tuberc 39, 9, 1939

STRAUB M Pathology of primary pulmonary tuberculosis, Med maandbl p 102 (Jan) 1947

STREEDT, H J Relationship between primary and adult tuberculosis, Brit J Tuberc 40 85, 1946

37. PULMONARY TUBERCULOSIS

SURGICAL ASPECTS

SINCE the turn of the century there has been general recognition of the value of local immobilization in the treatment of pulmonary tuberculosis and increasing use of surgery to enforce such immobilization and to close tuberculous cavities. In spite of the immense amount of interest in the subject and in spite of much experimentation to date no ideal surgical treatment generally applicable and offering predictable results has been devised. Hence the physician is confronted with multiple possibilities when he prescribes surgical treatment for tuberculosis.

While this book is devoted to diagnosis of chest disease the number of patients who present themselves to the physician in ordinary practice and who have had previously some kind of surgery for tuberculosis is sufficiently large to make brief descriptions of the various common surgical

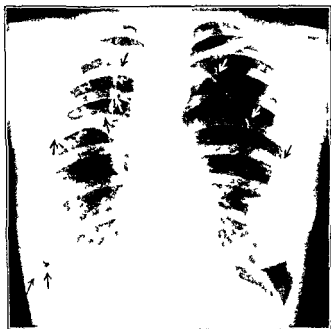
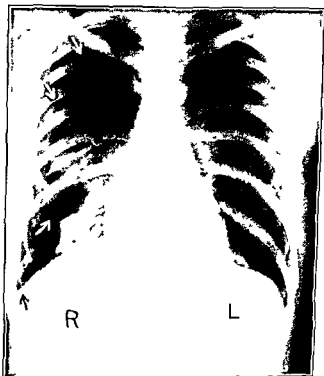


Fig. 101. Tuberculosis. (Left) Frontal view. (Right) Lateral view.

procedures of some value. Roentgen-ray films of the results of the various technics are appended.

Artificial Pneumothorax—The value of collapse in pulmonary tuberculosis was first recognized in the observation of patients in whom spontaneous pneumothorax had occurred, and artificial pneumothorax was thus naturally the first surgical means developed to induce collapse. It remains the simplest treatment, and the treatment of choice where there are no contra-indications.



- 1 Pulmonary tuberculosis
- 2 Right partial pneumothorax
- 3 Atelectasis of lower right lobe

The general technic of pneumothorax has been described under "Diagnostic pneumothorax" (see page 26). The procedure is fairly simple and is not subject to a great many possible complications. The needle should be kept close to the upper surface of the nearest rib to avoid the intercostal artery. Penetration of this artery, with resultant air embolism, is the greatest danger in the procedure. A close watch of the manometer, so that no air is injected until it shows the proper fluctuations with respiration, will obviate this hazard. The area for injection should be fluoroscoped

immediately prior to the operation with the patient upright and then in the lateral decubitus position affected side uppermost. The latter position is also, of course, the most satisfactory for actual operating procedure. The first injection should not exceed 200 cc of air. This should be followed immediately with further fluoroscopy. The pneumothorax is best visualized in the same position as for the operation. If the patient is then placed upright before the screen the air can be seen to move to the upper portion of the chest cavity unless prevented by adhesions.

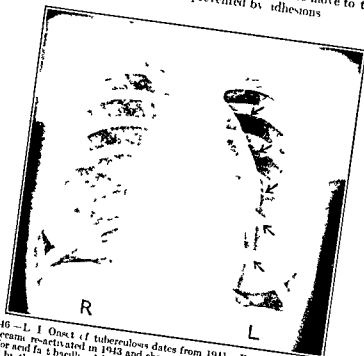
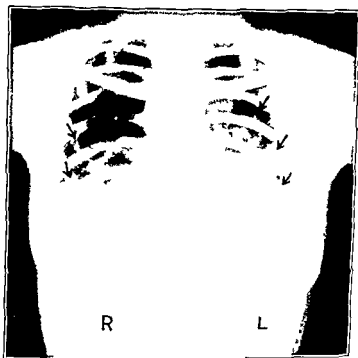


FIG. 146—L. I. Onset of tuberculosis dates from 1941. Patient improved but the disease became reactivated in 1943 and she entered the sanatorium. The sputum was positive for acid fast bacilli. A lesion in the left lung in addition to pneumothorax was suggested by the physical signs. This roentgenogram shows excellent collapse of the entire left lung. However, an uncollapsed cavity still exists between the two upper arrows. Some fluid is noted at the base of the left pleural cavity. The hilum of the right lung seems to be widened but this may represent a displacement of this lung by the collapsed left lung. Diagnosis—Left pneumothorax and tuberculosis.

Barring complications a refill should be made forty-eight hours after the first injection. Fluoroscopy is again a necessity. The procedure is the same as for the first injection except that a sharp needle can be used because the lung is no longer close to the chest wall. At the second injection 200 or 300 cc of air can be introduced. Manometer readings of pleural pressure should be taken after each 100 cc. In most instances small refills should be repeated at frequent intervals thereafter until the desired amount of compression is achieved because a large injection of air at one

operation sometimes makes it difficult for the thoracic organs to accommodate themselves to the altered pressure and space relationships.

Artificial pneumothorax is recommended both by its simplicity and safety. Unless spontaneous pneumothorax develops, or unless the air pocket stretches adhesions the operation should be painless. If the patient develops pronounced ill effects during the operation it can be stopped immediately and if necessary the air already injected can be removed.



sides.)

Diagnosis. Bilateral tuberculosis with bilateral pneumothorax.

The chief difficulty to the use of a pneumothorax is the adhesions which are frequently associated with any disease process of the lung and which may also be limited to the apices of the lung. It may also be limited to the apices of the lung—be so displaced as to prevent the achievement of collapse is achieved. The third limitation is the tendency of the injected air to collect at the apices of the lung—a disadvantage if the tuberculosis is primarily basal.

Intrapleural and Open Pneumolysis—Pneumolysis is a subsidiary technique to artificial pneumothorax. It may be undertaken whenever a satisfactory pneumothorax is prevented by adhesions which are yet not too numerous to make severance impractical.

Intrapleural pneumolysis is accomplished with the thoracoscope whose use has been earlier described with respect to diagnosis (see page 28). Cauterization of the adhesions may be through a second cannula or through the cannula used for the optical unit as in the unit designed by the author. The procedure is simple and easily controlled since it is performed under direct observation. Small blood vessels can be obliterated with the flat

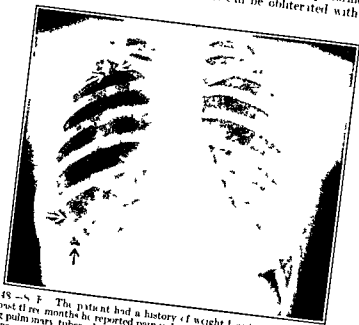


FIG. 148.—S. F. The patient had a history of weight loss totaling thirteen pounds for the past three months, he reported pain in both sides of his chest. He was diagnosed as having pulmonary tuberculosis in a routine mass roentgen ray study. Physical signs were dullness over the right upper lobe with a few rales. The roentgenograms taken on admission to the sanatorium showed a cavity in the right subapical area. A pneumothorax was done on this side. In this film the adhesion preventing satisfactory collapse is indicated by the upper arrows. The middle arrow indicates the edge of the partially collapsed lung, the lower a fluid level. Diagnosis—Chronic pulmonary tuberculosis, right pneumothorax with adhesions and serofibrinous pleurisy.

side of the cavity. It should be remembered, however, that a section of lung can be drawn into an adhesion of even moderate size, and therefore cauterization near the lung surface may produce spontaneous pneumothorax and the attendant possibility of pleural infection. Cauterization near the chest wall is, however, apt to be painful. This difficulty can be eliminated with a prior injection of novocaine through the cannula into the tissues of the chest wall. A high frequency current is suggested by Matson, which actually produces molecular disintegration, is a major development in the

use of this technic since it eliminates the smoke that may otherwise obscure the operator's view and since it seals lymphatic and blood vessels and thus minimizes the possibility of extension of the infection.

Intrapleural pneumolysis is a reasonably safe procedure in the hand of one familiar with the technic and may be undertaken whenever a few adhesions prevent a pneumothorax that would otherwise be effective. Open pneumolysis on the other hand is a more radical procedure and is indicated in those instances in which such procedure is indicated.

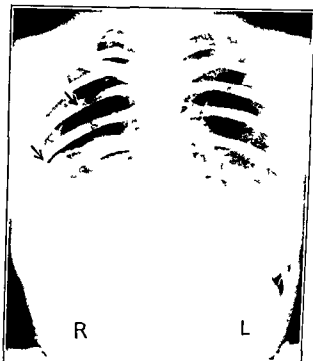


FIGURE 1. Intrapleural pneumolysis.
The arrows

point to the

area of the chest wall to be opened for intrapleural pneumolysis. With the chest wall laid open the actual procedure of severing adhesions is simple and fast. Large adhesions can be clamped with a hemostat before cutting to prevent hemorrhage which might otherwise be severe.

The risk of infection which accompanies large scale opening of the chest wall is minimized by the use of penicillin and streptomycin pre-operatively by the cleansing of the cavity with saline solution and more penicillin at operation and by daily post-operative drainage and further penicillin and streptomycin for a period of eight to ten days. Special care to prevent the

lung from expanding should be taken in the beginning during refills are advantageous.

It is possible that with the large scale use of antibiotics in operative procedure with consequent minimizing of infection opening of the chest wall will gradually become less awesome and open pneumolysis will become a more common operation. Certainly the results reported by Vidoppen recently indicate that the procedure is completely practical. However at the present time opening of the chest cavity with no other end than the severance of adhesions is not widely practiced.

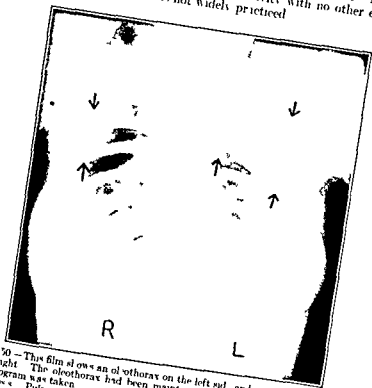


FIG. 150—This film shows an oleothorax on the left and an apical thoracic plastic on the right. The oleothorax had been maintained for eight years at the time this roentgenogram was taken.
Diagnosis: Pulmonary tuberculosis with oleothorax. Right partial thoracic plastic (right arm).

Oleothorax Oleothorax after a rather brilliant start has fallen into some disrepute and is not resorted to as much as formerly. Its chief advantage over artificial pneumothorax is that the collapse once established with oil maintains itself indefinitely without further attention. The technique is as follows: One or 2 cc. of the oil chosen paraffin is usually the oil of choice because of the irritating qualities of vegetable oil are introduced into the pleural space of the established pneumothorax to test for sensitivity. If there is no reaction 4 cc. of oil is added at the next pneumothorax refill. At each refill thereafter the amount is gradually

increased until there is sufficient oil in the space to maintain the desired degree of lung collapse. The patient is then free of the necessity for further injections. The procedure must be done cautiously and may require as long as three months for the injection of the required amount of oil.

The limitation of the procedure is obviously the amount of collapse that can be achieved by this method.

The development of a pleuro pulmonary fistula has been reported in the presence of an oleothorax and is a serious development. The entry of the oil into the lung may lead to fatal pneumonia. This may occur at any time after the introduction of the oil.

The most serious drawback, however, is due to the tendency of the pleura to form adhesions in the presence of any foreign matter. Thus it is frequently found that at termination of the oleothorax treatment the lung cannot re-expand. The temporary collapse becomes permanent and may require a major thoracoplasty.

Altogether the application of oleothorax in spite of its dramatic effect in individual instances is limited. For some time its use has been confined to those individuals in which frequent air refills are impossible because of distance from the physician.

Phrenicectomy and Phrenic Crush — This is another treatment which after wide application is being now used less and less by most thoracic surgeons. The operation is undertaken to paralyze one leaf of the diaphragm which then rises high enough into the chest cavity to compress the affected lung to some degree and by its relative immobility to provide some degree of rest.

At the present a phrenicectomy consists of the excision of 2 or 3 inches of the phrenic nerve and of the nerve to the subclavius as well as accessory fibers between the phrenic and cervical nerves which may otherwise provide a new circuit to the diaphragm. The more common operation, the phrenic crush, is usually effective in paralyzing the diaphragm for at least three months and in many instances diaphragmatic movement is permanently impaired.

In either case the operation begins with an incision about 2 inches in length parallel to the clavicle and directly above it. The sternocleidomastoid muscle is split longitudinally over the scalenus anticus muscle.

often the
ing the an

fibers are
impaired
t the rule

A phrenic crush has for some time been the treatment of choice in instances of unilateral basal tuberculosis and it has been used in other cases of tuberculosis when artificial pneumothorax was impractical. Recent interest in diaphragmatic function, however, has seriously threatened the prestige of the operation. In addition to the impairment of esophageal and gastric function which the organism tolerates well, it has been shown to inhibit in many patients the mechanism of cough. Cough in moderation

is the
that
of th
is use

Pneumothorax is the use of this method of collapsing the lung is reserved for those in whom pneumothorax is not indicated as a cause of pleural adhesions. The best results are obtained when the pneumothorax is performed on the side of the diseased lung. When

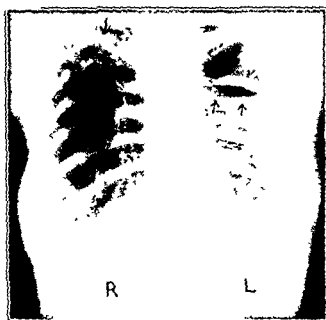


FIG. 151.—A. R. chest, forty-year-old female. Disease began in 1927 with moderate cough and fatigue. Diagnosis of tuberculosis was made in 1927 and pneumothorax treatment at that time was unsuccessful. Left pleurectomy was done in 1929. Extrapleural pneumothorax done in 1938 and the resulting fluid, blood and fat loculi. In 1940 the extrapleural pocket had been converted partially to an empyema. (See arrows.)

Diagnosis. Left pleurectomy, extrapleural pneumothorax and oleothorax. There is a cavity in the right upper lung. (See arrows.)

the air is introduced in the peritoneal cavity the diaphragm is forced upward much more on the side which is paralyzed by the pleuritic crust.

The method of introducing air into the peritoneum is simple. The skin near the umbilicus is washed with tincture of iodine followed by alcohol. Anesthesia is produced locally in a similar manner as in preparing the patient for artificial pneumothorax. A needle 5.2 cm. (gauge 19, length 2 inches) is used. The needle should have a relatively dull edge and is pushed into the peritoneal cavity. The bowel is rarely punctured as it slips away from the needle. The manometer readings are usually reverse

of the pleural readings and are not as accurate in expressing the abdominal pressure for obvious reasons.

When the manometer shows good oscillations 200 cc of air is introduced. If no reaction follows a few days later 500 to 1000 cc of air are introduced. Usually 1000 cc weekly are sufficient to keep the lung well compressed.

Banyai in a recent monograph states: "Pneumoperitoneum can be used with advantage in fibrocicous, caseous and ulcerative forms of tuberculosis. Exudative lesions of recent origin which frequently develop empyema when artificial pneumothorax is induced respond favorably to pneumoperitoneum treatment. Mason stated that tuberculous pneumonias demand pneumoperitoneum treatment alone if any form of relaxation therapy is considered applicable. Thin walled cavities are collapsible by pneumoperitoneum regardless of their location provided adhesions to the chest wall do not obviate their closure by pulmonary relaxation."

One should not hesitate to give artificial pneumoperitoneum treatment in upper lobe lesions for fear that relaxation of the intervening apparently

increased contractility will manifest itself in the relaxation of the diseased apical or subapical portions of the lung when pneumoperitoneum is well established. Furthermore incidental relaxation of the intervening healthy lung tissue is entirely harmless because as it was pointed out by Pinner no diffuse fibrosis or atelectasis follows the therapeutic relaxation of normal lung tissue.

The best prospects are cases where the lesion is a basal one or it is localized in the middle one third of the lung. Because the lower lobe is in direct contact with the diaphragm and it follows the respiratory excursions of this muscle it is moving much more than the upper lobe. Consequently one may anticipate a beneficial therapeutic influence from elevating the level and restricting the motion of the diaphragm by pneumoperitoneum in cases where the disease is localized in the lower lobe. (See Fig 43 page 95.)

Extrapleural Pneumothorax—This operation is still very highly regarded by competent authorities but most thoracic surgeons at present prefer radical surgery.

The operation is usually accomplished with a local anesthesia. The patient is placed upon the table with the area to be collapsed uppermost. An oblique incision is made in the skin above the center of the desired space. The muscles are divided and 2 or 3 inches of rib removed. Then first with the finger and then with an illuminated spatula the outer pleura is separated from the chest wall. This cavity may be made as large as desired. It has been suggested that it be one third larger than the size required for effective collapse. The space thus made may be filled with air, or more

is aspirated as well as saline solution. Pressure should be increased as the wound heals until the

desired amount of collapse is achieved. After the cavity is established refills need be no oftener than in intrapleural pneumothorax.

The procedure has attractive advantages. All procedures are performed under direct vision and the risk of accident especially lung perforation is minimal. Tuberculous infection of the area is rare and other infection can be minimized with pre-operative and post-operative use of penicillin and streptomycin. High pressures can be used and cavities that because of fibrosis, resist artificial pneumothorax can be closed. Refills are as easily

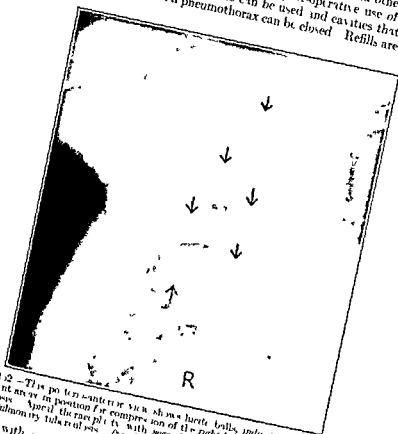


FIG. 12.—This postero-anterior view shows lucite balls, indicated by the circular translucent areas in position for compression of the right lung (see also Fig. 11). Apical the radiopacity with secondarily extrapleural cavity in lucite balls. Pulmonary tuberculosis (see Fig. 13).

made as with any other technique. Most important the collapse achieved is selective to a degree not found in any other technique the cavity size and shape is determined by the decalculation.

Extensive involvement contraindicates extrapleural pneumothorax of course. At the time of the operation it may be found that the pleura and chest wall are very densely adherent and the operation becomes too dangerous to be practical. Mediastinal shifting to the contralateral side may occasionally force the physician to discontinue the operation. (See Fig. 13, page 293.)

Plombage This procedure is in the same relationship to extrapleural pneumothorax as oleothorax is to artificial pneumothorax and relative advantages and disadvantages are similar.

The operation is begun as in extrapleural pneumothorax except that the cavity thus made is packed with paraffine or more recently with lucite (methyl methacrylate) balls. The aim as in oleothorax is the creation of a cavity that will remain without further attention as long as collapse is desired.



pl
ca

The disadvantages are those attendant upon any foreign substance under the skin: the possibility of irritation, localization point for infection and fistulization. Further and unique disadvantages are the tendency to bite of the lucite balls to migrate and the serious disability inherent in the necessity to full collapse in one or two operations. Because of this, mobile mediastinum is contra indication.

Cavity Drainage—Due to the insistent effort of Mondini, cavity drainage has become a recognized procedure in the treatment of tuberculosis. His technic of withdrawal of air from large cavities by aspiration offers a

FIG 154



14 15



11 12

For 154 - Apr 27 1940 Residual cavity 1 in at 1 ft ft receptivity. It is plants
 when as an oval shell with a lining well above and below the cavity. It is plants
 For 155 - Aug 27 1940 A small has been inserted into the cavity and 1 ft exposed
 atmosphere. No cavity can be seen.
 For 156 - Jan 6 1941 A skin flap has been inserted into the cavity using the pro-
 cess of Am. R. Tuberculosis. (S. p. man R. goes at 1 Dan 14 cur

For 155 - Aug 27 1940. A small insect was found on the plant. It is a planthopper. No other insects were found. A small insect was found on the plant. It is a planthopper. No other insects were found.

11-196-Jan 6 1941 A skin flap has been inserted into the cavity and left exposed
for approach. The cavity disappears at once. (S. pinn. R. goes at 11 Dan 14 cur
boy of Am. R. Tuberculosis)

Plombage This procedure is in the same relationship to extrapleural pneumothorax as oleothorax is to artificial pneumothorax and relative indications and disadvantages are similar.

The operation is begun as in extrapleural pneumothorax except that the cavity thus made is packed with paraffine or more recently with lucite (methyl methacrylate) balls. The aim as in oleothorax is the creation of a cavity that will remain without further attention as long as collapse is desired.



The disadvantages are those attendant upon any foreign substance under the skin: the possibility of irritation, localization point for infection and fistulization. Further, and unique disadvantages are the tendency to date of the lucite balls to migrate and the serious disability inherent in the necessity to full collapse in one or two operations. Because of this a mobile mediastinum is contra indication.

Cavity Drainage—Due to the insistent effort of Monaldi, cavity drainage has become a recognized procedure in the treatment of tuberculosis. His technique of withdrawal of air from large cavities by aspiration offers a

FIG. 154

FIG. 155

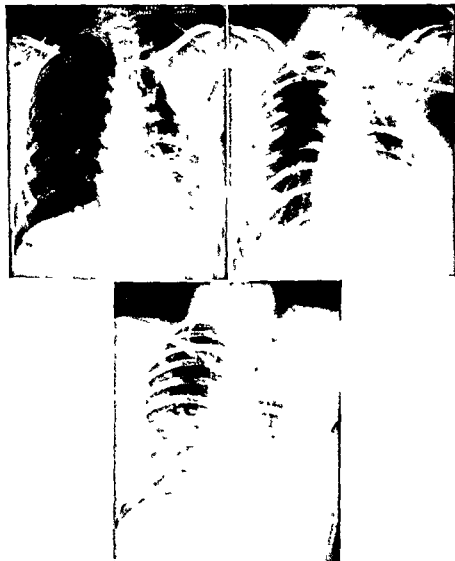


FIG. 156

FIG. 154—Apr. 22, 1940. Residual cavity beneath left thorax plate. It is plainly seen as an oval shadow extending well above and below the clavicle.

FIG. 155—Aug. 27, 1940. A wedge has been inserted into the cavity and left exposed to atmospheric pressure. No cavity can now be seen.

FIG. 156—Jan. 6, 1941. A skin flap has been inserted into the cavity, using the posterior approach. The cavity disappears at once. (Slipman, Rogers and Daniel, *Journal of American Review of Tuberculosis*.)

Plombage—This procedure is in the same relationship to extrapleural pneumothorax as oleothorax is to artificial pneumothorax and relative

(methyl methacrylate) balls. The aim as in oleothorax is the creation of a cavity that will remain without further attention as long as collapse is desired.



The disadvantages are those attendant upon any foreign substance under the skin: the possibility of irritation, localization point for infection and fistulization. Further and unique disadvantages are the tendency to date of the lucite balls to migrate and the serious disability inherent in the necessity to full collapse in one or two operations. Because of this a mobile mediastinum is contra indication.

Cavity Drainage—Due to the insistent effort of Monahan cavity drainage has become a recognized procedure in the treatment of tuberculosis. His technic of withdrawal of air from large cavities by aspiration offers a

FIG. 154



FIG. 155



FIG. 156

FIG. 154—Apr. 22, 1940. Residual cavity beneath left thoracoplasty. It is plainly seen as an oval shadow, extending well above and below the clavicle.

FIG. 155—Aug. 27, 1940. A needle has been inserted into the cavity and left exposed to atmospheric pressure. No cavity can now be seen.

FIG. 156—Jan. 6, 1941. A skin flap has been inserted into the cavity, using the parascapular approach. The cavity disappears at once. (Slipman, Rogers and Daniels, courtesy of *Am. Rev. Tuberc.* 1941.)

method of cavity reduction in which the risk of contamination of the chest wall and pleura is small enough to make the procedure feasible. It may or may not lead to permanent closure of the cavity. At the present time it is usually regarded as a pre-thoracoplasty technique; it probably deserves in some instances trial as an independent procedure. It is accomplished most efficiently, of course, with fluoroscopic visualization.

Eloesser, Rogers, Shipman and Kupka have varied the procedure and have also reported some good results. However, in most instances the



FIG. 157.—Cavity drainage by the Monaldi technique, showing the ruler tube in place at arrows.

Diagnosis. Pulmonary tuberculosis. Cavity drainage (Monaldi).

reduction of the cavity size has now become a procedure which precedes a thoracoplasty. Occasionally Eloesser's method of suturing a skin flap directly to the cavity wall has been helpful.

Thoracoplasty—Due in large measure to the effort of the late Max

procedure offers a reasonable chance of saving the patient's life. In such instances do occur and will continue to occur, and thoracoplasty will continue to be used, though to a lesser extent than at present, for some time.

Thoracoplasty is an operation which was first suggested by Brauer of Hamburg. The operation consists of a resection of one to eleven ribs as near the vertebral column as possible although it is customary to remove a portion of the transverse process of the vertebra also. The collapse of the chest wall is brought about by the resected ribs falling together and space between them becomes obliterated by the two ends uniting or if not a small bridge of periosteum forms, thus narrowing the chest to the degree according to the size of the ribs removed. The operation depends largely



FIG. 158.—This film taken with a Bucky diaphragm is of the same patient as in figure 157. It shows a right upper lobe thoracoplasty with the residual cavity outlined with iodized oil.

Diagnosis.—This residual cavity aspiration (Mondak).

on the degree of mobility of the ribs at their junction with the sternum. The amount of ribs removed indicates the type of operation. Most patients do unusually well with only a partial thoracoplasty and it was soon recognized that if only the upper portion of the lung was diseased it was more effective in collapsing the cavity and thus saving the lower or better portion of the lung. It is also noted that the mortality rate was markedly diminished so that now complete thoracoplasties even in several operations are reserved for the individuals who have an entire lung diseased and are otherwise in good condition.

The indications are the same whether it is an apical or complete thoracicoplasty the difference being only in the extent of the involvement. The dangers of thoracicoplasty are the development of pneumonia either in the opposite side or on the same side. If the operation is done rapidly and there is little or no shock and the collapse of the lung is not too great complications are less apt to happen.

At the present time the most important evidence to thoracic physicians and surgeons that a thoracicoplasty has been a failure and further treatment



is necessary is the presence of uncollapsed cavities. In order to recognize their (2) with a stratification which fail to respond by the posterior thoracicoplasty must be attacked by further operations in which more ribs are removed (anterolateral) and as many segments of the ribs be removed as the size of the cavity warrants. The newer type of operation as suggested by Semb (cf Swenson) has prevented

in a great measure, the so-called secondary operation for the closure of cavities.

As indicated thorico-plasty should be a treatment of last resort. As such it can and does save life. It may close cavities not amenable to other treatment and a speedy arrest of the tuberculous process is frequently obtained. With proper pre-operative preparation of the patient mortality rates are low.

Lobectomy and Pneumonectomy—Not many surgeons have reported using these obviously practical though desperate techniques in the treatment of tuberculosis. They are likely however to gradually assume a larger



the of

in the future

at operative

However

in the instances so far reported there is a tendency for relapse at a later time

The chief indications for a lobectomy are stenosis of a major bronchus not remediable by bronchoscopy or fibrotic cavities so thick walled as to be uncollapsible. It may also in the future be extended to cavitating tuberculous lesions confined to one lobe. The last indication is doubtful.

The indications are the same whether it is an apical or complete thoracoplasty the difference being only in the extent of the involvement. The dangers of thoracoplasty are the development of pneumonia either in the opposite side or on the same side. If the operation is done rapidly and there is little or no shock and the collapse of the lung is not too great complications are less apt to happen.

an



is necessary is the presence of uncollapsed cavities. In order to recognize their extent and to plan the operation, the patient should be examined with the thorax in the upright position. The cavities which fail to respond by the posterior thoracoplasty must be attacked by further operations in which more ribs are removed (anterolateral) and as many segments of the ribs be removed as the size of the cavity warrants. The newer type of operation as suggested by Semb of Sweden has prevented

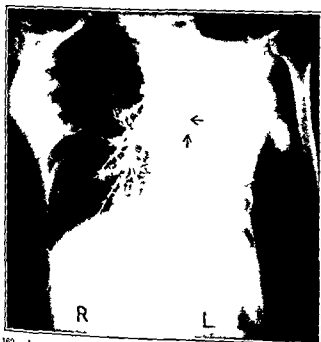


Fig 162—3
postoperative

lung " " pulmonary tuberculosis thoracoplasty (left)

left

because of the frequency of lesions undetectable by roentgenology which if located in fissures demand pneumonectomy when found upon operation to minimize the risk of further infection.

Pneumonectomy is a desperate remedy indicated by extensive unilateral destruction and contralateral infection or by stenosis of a major bronchus. If the patient survives the operation and post-operative period he will presumably have a better chance for recovery with major foci of infection removed in spite of some loss of vital capacity. As hope when all hope is gone the operation will probably establish itself securely in the armamentarium of the physiologist.



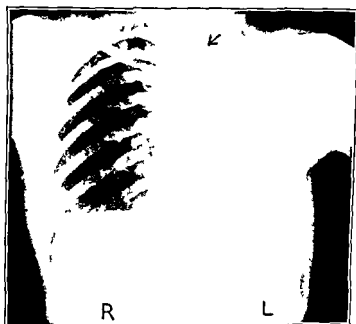


normal

Diagnosis was
lung

Pulmonary tuberculosis thoracoplasty (left) complete

Atelectasis, left



with expectoration

This roentgenogram shows almost complete opacity of the entire left lung. At arrow the trachea is markedly to the left.

Diagnosis—Left pulmonary tuberculosis with complete atelectasis.

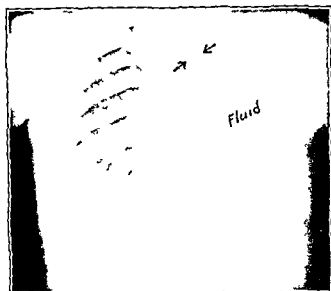


Fig 161 Same patient as shown in Fig 163 after talc pneumothorax. Talc stimulated to left (see arrow) left pleural cavity.

Diagnosis: Talc pneumothorax for tubercle sclerosis.

BIBLIOGRAPHY

GRANT, I. A. and ER, J. J. (1934) *British Medical Journal* (Chest) 133, p. 77.

PNEUMOTHORAX

ALLEN, I. V. and ELY, C. W. (1934) *Artificial pneumothorax* p. 140.

non-analytical
140
Whitney 32 1900
13 1870

Zellweger, G. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

CRANFORD, C. and ER, J. J. (1934) *Artificial pneumothorax* p. 140.

SINGER, J J " " " " " " " " " " " "

" " " " " " " " " " " "

pulmon " " " " " " " " " " " "

INTRAPLEURAL PNEUMOLYSIS

ANDERSON, D S and L. H. HARRIS " " " " " " " " " " " "

C

LITERATURE 19, 43, 1934

LOENNER, I Intrapleural pneumolysis, S Clin North America 6, 381, 1926

GLOYNE S R The pathology of pleural adhesions in pulmonary tuberculosis, Tubercle 7, 424, 1925-26

Goo

Hud

JACO

& Obst 34, 289, 1922

JACOBSEN, H

pneumothorax

Monatsh 18

33, 1931

33, 1931

OPEN PNEUMOLYSIS

SINGER, J J Thoracoscope in pulmonary diagnosis Am Rev Tuberc 10 67, 1924

VIDOFFEN, H F Wide-open intrapleural pneumolysis, Nord med 33, 110, 1917

OLEOTHORAX

GULLBRING

76, 238 1931

MOYER J H JR Oleothorax, Am Rev Tuberc 55, 223 1947

POLLOCK W C, and SKINNER R B Oleothorax therapy, J Thoracic Surg 3, 12, 1933

PHRENICECTOMY AND PHRENIC CRUSH

" "

O BRIEN, F J PHRENIC NERVE OPERATIONS in Lung Cancer, J Clin Oncol 9, 600, 1930

STERTZ Kunstliche Zwerchfellumlung bei schweren chronischen einseitigen Lungenerkrankungen, Deutsche Med Wochenschr 37, 2224, 1911

PNEUMOPERITONEUM

ARNOLD, L Intrapertoneal injection of oxygen in the treatment of acute diffuse peritonitis, J von chir 8, 411, 1912

No. 13, 14

1 34 7 44 8 6 20=

4

WELLS, I. S. Difficulties of the plastic and unclogging cavities. J. Theoret. Surg. 6:4-10, 1937.

LOBECTOMY AND PNEUMONECTOMY

BRUNNER A. The surgical aspect of bronchial tuberculosis. Schweiz. Zt. f. d. Tuberk. 4: 218, 1937.

32 1945

CHURCHILL, F. D. and KLOPFER, R. Lobectomy for pulmonary tuberculosis. Ann Surg 117: 611, 1943.

CLETT O T and LUCKEY C A. Letter my for pulmonary tuberculosis. Pre
Staff Meet. M. 11-19-68 1114

a man stuck in. Travelers

of tuberculi is introduced by

next part of case 3. The review

THE UNIVERSITY OF CHICAGO

Surg 15:384, 1986

Schwartz P C. Indications for lobectomy and pneumonectomy in pulmonary tuberculosis. *Ann Surg* 112:201 1910

SWEET R H Lect on alpacum met in th treatment f the nars t the
thess J Th ruc Surg 15 373 1110

TH RYAN T F JR at Adams W I file no t i filing to t f r j do nary
t brcastals Sir (M & O) t 75 312 1112

38. BENIGN TUMORS OF THE LUNG

BENIGN tumors of the lung are ordinarily divided into two groups. Adenomas found in or near the bronchial tree are relatively common. They are estimated on the basis of various statistical studies to comprise between 5 and 15 per cent of all pulmonary neoplasms. The other group consisting of fibromas, chondromas, osteomas, myomas, endotheliomas, etc. are found infrequently in the lungs.

Etiology—There is no agreement as to the etiology of bronchial adenomas. Because they at times microscopically resemble fetal lung tissue and because of an apparent tendency to occur in conjunction with congenital anomalies of the bronchial tree. Womack and Graham advance the theory that they represent infolges that failed to develop normally. Brack emphasizes the possibility of their origin in bronchial mucous glands and Fried recognizes this origin and also a more common origin from the basal cells of the bronchi—the origin also of bronchogenic carcinoma. It seems possible that the bronchial adenoma may arise from any of these sources.

The other benign tumors of the pulmonary system are usually found in the alveolar tissue although in some instances reported a bronchial origin is a possibility. It is likely in spite of the advanced age at which a few patients with this condition have come to the attention of physicians that these tumors are congenital in origin.

Pathology—Adenomas are thought to be of very slow growth. They usually begin as wart like protruberances on the bronchi originating either within the mucous membrane or within the bronchus itself. Ordinarily the adenoma consists of the growth within the bronchus and its attachment but sometimes one is encountered with its main body on the exterior of the bronchial wall.

Most adenomas gradually become pedunculated and grow pointing toward the trachea. Some of them seem to grow both directions from the attachment to the bronchus. They are usually rather regularly oval in

In color adenomas vary from white to pink and are usually smooth and slick. In consistency they tend to be quite firm. They often contain an extraordinary number of small blood vessels and may bleed profusely when irritated.

Sooner or later an adenoma will completely block off the bronchus and atelectasis follows. Unless there is surgical intervention at this point the lung will atrophy and infection may set in. Empyema is a frequent sequel to this condition and has been the direct cause of the patient's death.

Occasionally adenomas have been reported that have eroded into the bronchus and very rarely, and most inexplicably, growths that are microscopically typically adenomatous will metastasize. Because of such occasional metastasis Graham and Womack feel that all adenomas are potentially malignant but there is still considerable debate among pathol-

regards as to whether a true adenoma can ever become definitely malignant.

In the other benign tumors the information as to pathology is scant. Growth is apparently very slow and most of them remain small. They are ordinarily discovered by roentgenologic examination for other causes.

degeneration, and it is quite possible that in the lung such a process is also possible. No instances have come to the author's attention, however, of such degeneration.

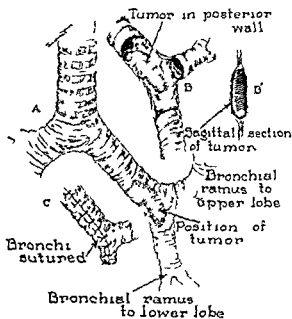


FIG. 16a. Bronchial tumor. (From *Diseases of the Chest*, July-August, 1937, By A. Goldman.)

Clinical Symptoms. Before atelectasis and infection develops, an adenoma will produce few symptoms. Hemoptysis is the most common indication of abnormality when the tumor lies over a large pulmonary vein; such hemoptysis may be severe. Dyspnea is common with degree depending upon the amount of lung tissue blocked off by the tumor. After atelectasis takes place and if infection develops, the patient will present fever and the usual symptoms of sepsis.

The rarity of parenchymal tumors makes the definition of a pattern of symptoms impossible. The endothelioma of Edwards presented symptoms that were purely mechanical: tightness in the chest, occasional dry cough, some hemoptyses, etc. Such symptoms are entirely dependent on size and

adenoma. When there is a complete block of the bronchi the sharp angularity of the interrupted iodized oil will indicate adenoma.

The other benign tumors will be seen roentgenologically as more or less rounded shadows. Their outlines will usually be sharp. When such tumors have ulcerated they will be indistinguishable roentgenologically from lung abscesses.

simple bronchoscopy should always be undertaken when adenoma is suspected. Caution should be exercised in the operation, however, because if the growth lies over a large pulmonary vein a very slight injury can cause a severe hemorrhage.

Bronchoscopy is of course little use in the diagnosis of those benign growths that lie beyond its field of vision.

Laboratory Tests—Cytological examination of a tumor specimen is always necessary to definite diagnosis. It is usually very simple to obtain biopsy specimens of suspected adenomatous growths, but other benign growths often offer difficulty. Whether or not the radical techniques of securing specimens of tissue for study are resorted to will depend upon individual factors in the case.

Differential Diagnosis—Adenomas, because they are comparatively small and silent and yet produce widespread pathologic disturbances, are frequently incorrectly diagnosed. They are most often mistaken for tuberculosis, recurrent pneumonia and carcinoma. Not infrequently conditions secondary to the adenoma—bronchiectasis and emphysema will obscure the growth itself in diagnosis.

The other tumors of the lung do not suggest such a wide variety of alternatives in diagnosis, but differentiation is apt to be more difficult within a limited range of possibilities. The most important differentiation to be made is that of malignancy, since the roentgenologic shadow may resemble the shadow of carcinoma. In other instances it may resemble a calcified tuberculous lesion. If the tumor ulcerates it will look and act very much like a lung abscess.

Tuberculosis—Tuberculosis will usually be suspected where hemoptysis occurs. Not infrequently the complications of an adenoma will make it difficult to eliminate tuberculosis roentgenologically. However the patient's history may be suggestive. In any patient suspected of tuberculosis in which no acid fast bacilli can be discovered bronchoscopy should be done; this will reveal the adenoma if one is present. (See Fig 104, page 207.)

Carcinoma—On the basis of symptoms, physical signs and even roentgenograms it may be very difficult to distinguish an adenoma from early carcinoma of the bronchus. Where the adenoma produces hemoptysis carcinoma will be particularly apt to suggest itself. Differentiation will depend on bronchoscopic examination, specimens for microscopic examination can be secured and will permit a conclusive diagnosis. (See Fig 81, page 163.)

Other benign growths, when not infected, can often be distinguished from carcinomas roentgenologically by their sharp edges. The absence of clinical

symptoms will suggest benignity. However the examiner should establish the benignity of such a growth beyond a reasonable shadow of a doubt and as soon as possible. If any doubt remains after the usual diagnostic technics have been exhausted the procedure except in unusual cases should be the removal of the suspicious growth by operation.

Bronchiectasis Bronchiectasis often results when an adenoma only partially blocks a bronchus. It may completely overshadow the tumor that causes it. However normal procedure in the presence of bronchiectasis is a roentgenologic examination after the injection of iodized oil. With this technic the tumor will be easily noted. It will of course also be visible if a bronchoscopic examination is undertaken. (See Fig 75, page 157.)

Empyema—This is also an instance of a secondary condition overshadowing a primary one. There are numerous reports in medical literature of prolonged treatment for an empyema before the original cause of the infection—an adenoma in a bronchus—was discovered. However the failure of the lung to re expand after the aspiration of the fluid as seen roentgenologically will suggest a blocked bronchus. bronchoscopy will reveal the block as an adenoma.

Calcified Abscess As has been suggested earlier in certain instances carcinoma, benign growths and calcified lung abscess when they are located completely within the parenchyma are impossible to identify short of punch biopsy or exploratory operation. If the examiner is perfectly sure that the lesion is not malignant he may in the absence of symptoms feel

ulcer
shallow
of the
tumor
ly
a

tumor will be discovered when open drainage is instituted. Biopsy will identify the kind of tumor. (See Fig 106 page 209.)

BIBLIOGRAPHY

- ADAMS, R. A. Discussion of symposium on bronchial tumors. *J Thoracic Surg* 14 115 1945
- ADAMS, W. E. STEINER, P. F. and BLOCH, R. G. Malignant adenoma of lung carcinoma-like tumors with long clinical course. *Surgery* 11 503 1942
- ANDERSON, W. M. Bronchial adenoma with metastasis to liver. *J Thoracic Surg* 12 351 (April) 1942
- BELL, E. T. Textbook of Pathology 6th Ed. Philadelphia: Lea & Febiger 1947
- BIGGER, I. A. Discussion of symposium on bronchial tumors. *J Thoracic Surg* 14 120 1945
- BROCK, R. C. Pulmonary new growths: pathology, diagnosis and treatment. *Lancet* 2 1041 and 1103 1938
- reference to bronchial adenoma
t polypoid bron
pulmonary re

39. CANCER OF THE LUNG

the future is certain to increase. The chief limitation to the technic so far encountered is the failure to diagnose the condition before extension of the disease has made surgery hopeless. There is perhaps no other condition where increased accuracy of early diagnosis offers so much possibility of concern only to

Many patients are asymptomatic until metastasis has made the condition hopeless but many more if carcinoma of the lungs is considered when symptoms are first noted can be saved.

We shall not in this chapter devote space to sarcoma of the lungs. In recent years more and more of the sarcomas reported in the past have been identified as small celled carcinomas and true primary sarcoma of the lung is now recognized as a rare condition. It is impossible to distinguish the condition from carcinoma except by biopsy.

Etiology
The incidence of lung cancer has increased in the last few years. It has been reported at various times from Montefiore, Barnes, Brompton and St. Luke's hospitals between 3 and 4 per cent of the patients with bronchogenic carcinoma were under thirty years of age. It has been rarely encountered in children.

Statistics do not confirm any of the theories advanced for this disproportionate incidence. The factors that most likely bear upon it are the greater susceptibility of males to all forms of pulmonary irritation and possibly the greater susceptibility of females to carcinoma of the generative organs. A sidelight to these statistics of some interest is the fact that one type of bronchogenic carcinoma, the adenocarcinoma, claims almost as many women victims as men.

Most authorities are by now agreed that some form of irritation is always an etiologic factor in cancer. In many cases and perhaps in all the irritation is chemical in nature. In the now famous Schneeberger Pulmonary Cancer studies a direct chemical agent as the irritant factor is a certainty. Moller in 1924 and later Murphy and Sturm demonstrated that primary lung tumors could be induced experimentally by the application of coal tar cutaneously. This together with the apparent marked increase in lung cancer incidence (which most authorities feel is illusory) has

led to the investigation of the hydrocarbons in the air of a modern community as a causative agent. These studies have been non-productive in demonstrating a direct causal relationship.

The possibility of micro-organisms as causative agents has not been neglected. This line of research has been enormously stimulated by the demonstration by Fibiger that rats following persistent ingestion of *Spiroplera neoplastica* developed cancer of the stomach. While it is certain

histories has discounted the suggestion that influenza is a causative factor.

In a small number of cases carcinoma has followed chest trauma. It is improbable however that trauma by itself is sufficient to cause cancer unless the tissue is at the time in a precancerous stage unless the regenerative cells are already over active due to chronic irritation. A few cases

some other of the dust-induced

This situation is too rare however. Asthma most often follows bronchitis when there is a history of previous lung abnormality at all but it is quite possible that such bronchitis is due to carcinoma and not the reverse.

As a matter of fact rather exhaustive studies of case histories in recent years have rather thoroughly demonstrated that there is no pathologic history that *per se* predetermines to carcinoma of the lung. It seems most likely that many kinds of irritants if they are chronic can lead to carcinoma.

Investigators have also been much interested in the second factor in the development of cancer—the question of natural resistance. Results in this direction have been less tangible although transplantation studies indicate that bodily resistance is a very real factor in the genesis of cancer. The experiments with tar application to animals has also been significant to this question. In the experiments of Murphy and Sturm in which bronchogenic cancer was induced by cutaneous application of coal tar their conclusion was not that the inhalation of tar caused the cancer but that the application of the tar to the skin of the animals weakened their normal resistance to cancerous growth generally and that specific irritants in their customary environment were the direct etiological factors. A search is still underway at the present time for a bodily hormone which would be the control factor in tissue repair and hence a factor in the causation of cancer.

Pathology—Cytologically carcinoma of the lung is ordinarily broken down into three groups. The author however prefers the division of Fried into five groups since it is possible that the oat cell, round cell and basal cell carcinoma lumped by most authors as undifferentiated cell carcinoma may offer slightly different prognosis as well as cell-structure difference.

The squamous cell carcinoma is the most common and includes about 40 per cent of all bronchogenic cancer. The most characteristic feature microscopically of this type of tumor is the cluster of cells surrounded by bands of fibrous tissue. There is no definite arrangement and the individual

and cells vary greatly in appearance. They are typically quite large and often polynuclear. It is this kind of tumor that shows a pearl arrangement not infrequently due to cornified epithelium.

Adenocarcinomas make up about 10 to 20 per cent of all bronchogenic

Oat cell carcinomas have been frequently described since Bernard first called attention to them in 1926. They present a rather uniform appearance of small oval or spindle cells usually lying more or less in one direction. This kind of carcinoma is vigorously invasive. Its victims are almost exclusively males.

The cells composing the basal cell epithelioma contain much cytoplasm and their nuclei are vesicular. They usually lie in strands two or three cells in width which are often in the form of whorls. There is ordinarily rather a lot of fatty tissue both within and without the cell bodies. There will be little uniformity in gross appearance.

The small round-celled or medullary tumor will contain cells of little individual variation but which lie in irregular groups. This tumor is particularly likely to metastasize to the liver in addition to blood vessel metastasis. However it does not ordinarily invade the nervous system.

A bronchogenic tumor is seldom uniform in its cell structure but in practically every case one cell type or another is sufficiently predominant to characterize the growth. As more information is accumulated the author feels that the above five-group division if generally used will perhaps provide information bearing on operability of carcinoma of the lung.

Fried and others have rather conclusively demonstrated that all these cell types have their origin in the somatic cells that line the bronchi since the more highly-differentiated cells of the body are fixed in their characteristics and since somatic cells are responsible for all the regeneration that takes place in the bronchi and lungs.

In present thought the mechanism of cancer formation begins with a particular group of undifferentiated cells being required to make a particular kind of tissue repair repeatedly because of some kind of chronic irritation mechanical chemical or bacterial. The cancer may actually start either by a group of such cells becoming themselves diseased and anarchic or by a systemic bodily imbalance of some kind developing as I have phrases it. It will be noted that these two possibilities are not mutually exclusive.

The great majority of bronchogenic carcinomas originate close to the hilus although rarely they may be found confined to the parenchymal more direct line for irritating. The growth is

ning of the dis-
character. As

it advances a true tumor body appears. Surrounding parenchyma may be invaded by nodules which as they enlarge join the main body of the tumor isolating bits of other tissue or by a more diffuse speckling of such parenchyma a miliary invasion. A large tumor will frequently undergo necrosis in its interior.

A carcinoma of the lung tends to be grayish white, often rather translucent in appearance. An adenocarcinoma is often streaked with yellow mucus. Sometimes blood and blood vessels will give the growth a pinkish appearance. Recently occluded blood vessels or other types of tissue are sometimes seen imbedded in the mass. Nodules lying outside the periphery are common and sometimes the entire lobe will be dotted with grayish miliary metastases. Where the parent growth lies away from the bronchial tree in the parenchyma of the lung it tends to be rounded or lobulated.

Because bronchogenic carcinoma tends to be silent until complications develop the rate of growth is a matter for speculation. In certain cases minor symptoms perhaps attributable to carcinoma have been found to precede the classic symptoms of malignancy for long periods of time. In general however the time elapsing between the onset of symptoms and the death of the patient is apt to be short. The out-cult variety particularly moves swiftly.

Early metastasis is a common feature of most carcinomas although Fried clumps the basal cell epithelioma as an exception. Adjacent lymph nodes are almost invariable sites for these metastases and for this reason surgical procedure includes removal of all such nodes. In later stages metastasis to the pleura is common and not infrequently the ribs will be affected. Erosion of ribs is particularly likely in tumors in the apex of the lungs. All bronchogenic carcinomas likewise with the exception of the basal cell type noted above metastasize through the blood stream. Fried thinks the preferential location of such metastasis depends on the type of tumor. The most frequent sites of such metastasis are the liver, adrenals, the kidneys, the skeleton and distant glands such as the pancreas. In addition carcinoma of the lung is more apt than any other malignant growth to metastasize to the brain.

As the tumor grows the area of cardiac dullness will be shifted to the affected side due to atelectasis. If the tumor has achieved any size chest movement on the affected side will be limited and frequently that side will be visibly depressed as compared with the other side.

fluid producing conditions.

Frequently the area of cardiac dullness will be shifted to the affected side due to atelectasis.

If the tumor has achieved any size chest movement on the affected side will be limited and frequently that side will be visibly depressed as com-

pared to the unaffected side. Unilateral decreased mobility should always suggest carcinoma in patients past fifty years of age.

Breath sounds will ordinarily be diminished and rales are frequent. The latter however tend to appear and disappear during the course of the disease. Occasionally when the tumor surrounds but does not obstruct a large bronchus markedly increased breath sounds similar to those of pneumonic consolidation will be heard.

In general the physical signs elicitable in tumor of the lung are indefinite and will be of less help in the diagnosis of carcinoma than they are in other diseases. Whenever the physical examination of a patient shows signs

the most frequent symptom with sputum pain and dyspnea present in more than half the cases analyzed and emaciation in almost half.

The injection of iodized oil has been found to be very helpful in roentgen ray
 chias
 susp

always present when a tumor is located near a major bronchus.

For tumors which lie entirely within the parenchyma of the lung fluoroscopic procedures are vital to diagnosis. Such tumors are usually round although they may be lobulated. A hazy infiltrative area most often

Sometimes an inflammatory process may almost completely obscure the primary tumor. In other cases where the tumor has broken down the roentgen ray appearance is that of an abscess of the lung.

In a large percentage of patients with carcinoma of the lung metastasis to mediastinal lymph nodes will already have taken place when they are

answer to the
 super imposed

When the tumor is present in or near the hilus symptoms may be very early. Frequently hemoptysis to a small degree appears very early due to the erosion of the tumor into the small blood vessels within the bronchus. Cough will almost always be present. Sometimes a peculiar soft hacking

cough will be noted by the physician in a patient who has denied that he has a cough. Sputum in early cancer is usually creamy white, mucoid and colorless.

Pain is a very common symptom. In the early stages of the disease it is usually a dull ache at the site of the lesion. Dyspnea occurs when bronchial obstruction seriously diminishes lung volume.

In later stages of growth the symptoms become more pronounced. In many instances the resultant bronchiectasis will symptomatically overshadow the tumor itself. The sputum will be purulent with a foul odor. The classic symptoms of malignancy—emaciation, rapid loss of weight, night sweats—appear ordinarily quite late in bronchogenic carcinoma and sometimes do not appear at all. Dilated superficial veins, cyanosis, edema and other symptoms of pressure within the thorax are of course dependent upon the location of the growth. In the terminal stages a tumor located near the hilus will erode into the mediastinum and cause severe pain, either continuous or stabbing, by its action upon the nerves within that structure. Hoarseness is not infrequent as a terminal result when the recurrent laryngeal nerves are invaded.

Apical lung tumors present a special body of symptoms in most cases, although it is generally agreed that their location is the cause for the superficial similarity within the group and that there is no advantage in trying to make a special category for them. Even quite a small tumor within this area can cause intense pain because of nerve and bone erosion and the

operation. Sometimes a dull pain at the site of the lesion, before mentioned, will indicate an abnormal process, but it is seldom severe enough to seriously alarm the victim in the early stages. Not infrequently, on the other hand, the presenting symptoms are those accompanying metastasis in some other part of the body.

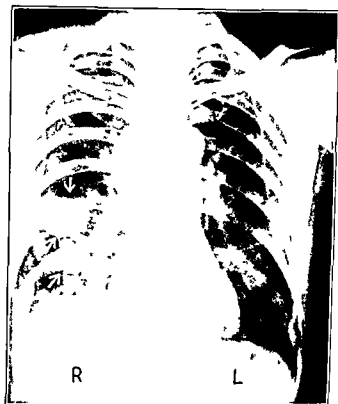
Roentgen ray Findings. The roentgen ray appearance of carcinoma of the lung are divided into two categories, the appearances of the tumor itself and the resultant complications within the thoracic cage. In many cases the first roentgenologic sign of a tumor is that of obstructive emphysema due to a partially constricted bronchus. As the obstruction becomes complete, atelectasis develops and a shadow, varying in size and density with the amount of atelectasis, replaces the lightened area of emphysema. Sometimes the atelectatic shadow is difficult to distinguish from the shadow of the tumor itself. Other signs of atelectasis, such as the displacement of the mediastinum and heart toward the affected side and a raised diaphragm, will also then be noted.

Sometimes the carcinoma itself will be visible as a hazy, infiltrated area in the lung much like the appearance in tuberculosis. When it is seen as a solid shadow, it will tend to be triangular in shape. It will usually extend out from the hilus or large bronchus. Atelectasis in adjacent tissue may make the tumor appear larger than it really is. If the bronchus is not obstructed, a hazy halo of infiltrated parenchyma will be seen around

the tumor mass. Small nodules of tumor growth frequently lie outside the main body.

Careful rotation of the patient behind the fluoroscopic screen will often assist shadows dissolve.

163, 90, page 182, 107,



Bronchoscopy—Bronchoscopy is perhaps the most important single method of lung tumor diagnosis since it not only permits a direct view of the growth in many instances but also oftentimes permits the taking of biopsy specimens. In a large percentage of instances the tumor itself will be visible bronchoscopically, where it is not in the field of vision of the bron-

obstruction

A specimen for biopsy should be of course taken of any suspect tissue. Where no such tissue is visible, scraping in the area will sometimes reveal cancer cells.

During bronchoscopy the examiner should carefully note the amount of uninvolved bronchus above the tumor. This information is of importance when the feasibility of surgical removal of a lobe or lung is considered.

Thoracentesis and Thoracoscopy—In addition to their diagnostic usefulness, these procedures are oftentimes also helpful in determining operability. Ordinarily the question of whether or not they should be used will be determined by roentgenologic studies.

Pleural fluid when present should be studied by the Mandelbaum method for carcinoma cells. However, in many instances carcinoma of the lung may indirectly produce a pleural fluid which does not contain evidence of its origin; a negative finding is not conclusive.

By thoracoscopy one can see the pleural adhesions so often associated with carcinoma. These are often extremely dense. Sometimes carcinomatous nodules will be seen on the pleural surface of the lung. Biopsy specimens of these nodules can be taken. The pleura should also be closely inspected for nodules or other indications of carcinoma that would be a contra-indication to operation. In many instances it will be possible to see whether or not mediastinal lymph nodes are swollen or carcinomatous.

Laboratory Tests—It is not within the scope of this work to detail the complex microscopic appearances of normal and abnormal cell structures. Suffice it to say that the physician should make every effort to confirm a tentative diagnosis of carcinoma of the lung by laboratory tests of specimens. The most valuable source for such biopsy material is of course the biopsy of the tumor itself. When such a specimen is not available, however, confirmation can sometimes be had by examination of sputum or pleural fluid or bronchial scrapings. In some instances diagnosis has been possible by microscopic examination of superficial lymph nodes, particularly supraclavicular nodes. Where these procedures fail the physician still has

is not a procedure of choice, since infection and metastasis to the pleura have been reported following the aspiration, but it should be used when certain diagnosis is impossible by other means.

The study of sputum secured bronchoscopically is also a vital part of the recent interest in bronchoscopic diagnosis of carcinoma. The technique is quite simple. The secretion should be secured if at all possible from the larger bronchi, the most likely area for malignancy. Smears should be

of secretion studies where as such diagnosis was possible on the basis of

other evidence in only 31 of the cases. This technic when more widely applied should be of inestimable benefit to the control of a disease where early diagnosis is so necessary.

Differential Diagnosis—As must be evident from the foregoing discussion in many instances a most thorough going examination is necessary to distinguish bronchogenic carcinoma from other pulmonary abnormalities. The most likely confusion occurs in distinguishing carcinoma from tuberculosis and from pulmonary abscess. Fungus disease may also cause much

will make the

mistaken for

primary bronchogenic carcinoma. They most commonly appear as numerous small irregular densities scattered over a wide area and often bilateral. Occasionally, however, there may be only a single metastasis visible roentgenologically. Unless the primary cancer elsewhere is known it is impossible to distinguish a single secondary tumor from a primary one except by microscopic study.

Tuberculosis The roentgenologic appearance of early invasive carcinoma is sometimes strikingly similar to that of tuberculosis and the physical signs and symptoms may also be similar. Greatly enlarged mediastinal

tuberculosis have been lost because of the delay occasioned by repeated

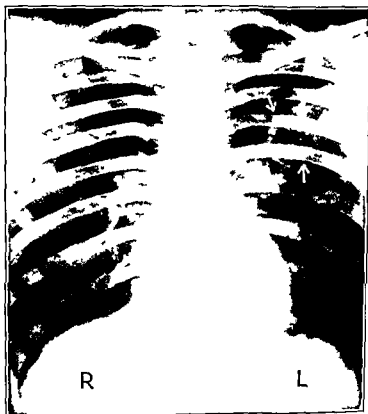
diagnosis of carcinoma may
due to the invasive process
differentiation is impossible

diagnosis of carcinoma
always be made
tissue for biopsy
(4)
the shadow on the

roentgenogram although it not infrequently will present the peculiar

frequent attacks of pneumonia even though such attacks conform to a classic pattern for that disease should suggest carcinoma (See Fig 142 page 281)

Silicosis—This disease is well enough known to be suggested whenever the patient's history includes a long period of exposure to silicious dusts. Although roentgenologic appearances may be similar to those of carcinoma the symptoms of silicosis are almost always of a much longer duration than



the symptoms of carcinoma. It is however no longer a rarity to find silicosis and carcinoma coexistent. Unless bronchoscopic examination

sions are
variably

demonstrate involvement elsewhere, usually in the long bones. Skin lesions are common. Since sarcoidosis commonly involves lymph nodes a biopsy of any enlarged, superficial node palpated is the simplest method of differentiation. A patient with sarcoidosis may otherwise be in the best of health.

Calcified Lesion in the Lung—The author and Dr. F. A. Graham reported three cases encountered at Barnes Hospital of old calcified lesions in the lung that were roentgenologically and symptomatically indistinguishable from tumors. A review of these cases in the light of new knowledge of



FIG. 170. Lateral brain scan same patient as in Figure 164. The dense mass is accentuated by this technique. (See arrows.) (A. C. Gillman, St. Louis, American Review of Tuberculosis, March 1948.)

Diagnosis. Arteriovenous fistula of the lung.

thoracic diagnosis still indicates no reasonable way of differentiation short of diagnostic puncture or exploratory operation. (See Fig. 104, page 297.)

Histiocytosis. As remarked earlier, it is sometimes difficult to exactly demarcate a tumor from the histiocytosis it produces. Likewise it may be difficult to tell whether or not the histiocytosis is due to a tumor. Ordinarily

the condition when due to other cause, will arise abruptly with rather severe symptoms of pain cough and bloody sputum. The history of a recent lung operation or foreign body inhaled will be significant. Broncho-copy will of course immediately distinguish the conditions. (See Fig 163, page 304.)

Benign Growths Carcinoma may occasionally be confused with a cyst or benign tumor. It should be preserved as a new growth of the major bronchus.

Microscopically secured biopsy specimens. In almost all cases of growths in the parenchyma a careful roentgenologic study will usually suggest a growth to be invasive or non-invasive. Whenever any doubt remains after a thorough roentgenologic examination the physician may resort to punch biopsy. (See Fig 166 page 312 Fig 169 page 326 and Fig 170 page 327.)

BIBLIOGRAPHY

- ALTGAUZEN A Y. Diagnosis of malignant growths from fresh smears. *Am Rev Synt Med* 4 148 1946
- BARNARD W G. The nature of the oat-celled sarcoma of the mediastinum. *J Pathol & Bact* 296 241 1926
- BEARD H H, HALPERIN B and LIBERT S A. Effect of intraperitoneal injection of malignant urine extracts in normal and hypophysectomized rats. *Science* 105 475 1947
- FRIED B M. Primary Carcinoma of the Lung. Baltimore: Williams & Wilkins 1932
- . Bronchiogenic carcinoma: study of 152 autopsied cases. *Acta Internat Union against Cancer* 3 153 1938
- FRIED B M. Bronchiogenic carcinoma and Adenoma. Baltimore: Williams & Wilkins C 1948
- FRIESEL L F and KNOX I C. Primary carcinoma of lung. *Am J Cancer* 30 219 1937
- HERBERT P A and CLARK L H. Bronchiogenic carcinoma diagnosed by cytologic study of bronchoscopically removed secretions. *J A M A* 130 1006 1946

40. SARCOIDOSIS (BOECK'S DISEASE)

SARCOIDOSIS was originally thought to be a cutaneous condition; it is now known to be systemic. In a large majority of patients with the disease there are pulmonary and lymph node manifestations. The spleen, liver, intestinal tract and kidneys are also involved with some frequency.

Sarcoidosis is seldom acute in its early stages. It ordinarily pursues a rather lengthy course with remissions in one part of the body taking place as new lesions appear elsewhere. In most instances sarcoidosis does not actually cause the death of the patient. Some form of tuberculosis is perhaps the most common cause of death. Right-sided heart failure due to extensive pulmonary fibrosis is not uncommon.

Etiology — Most investigators in this field now believe that sarcoidosis is a noncaseating tuberculosis, although tubercle bacilli are seldom demonstrable. Pinner's explanation for this failure is the most convincing, he

probable that the bacilli in question are of a low degree of virulence. The most puzzling etiological question is why certain hosts should react in such a non-typical manner to acid fast bacilli. The recent experiments with killed tubercle bacilli in which such bacilli produced typical sarcoids on patients with Hodgkin's disease may furnish a clue to investigators of Boeck's disease.

Pathology The sarcoid begins in soft tissue as a small area of inflam

ally, necrosis does not take place to an appreciable degree.

Sarcoids are small, round and sharply defined. Development is slow. When they are subcutaneous they range from a dark red to brown or purple. When they do not lie on the surface of an organ they are hard, rubber and spheroid.

Resolution of the lesion may be by resorption over a period of years. More commonly the sarcoid is encapsulated or replaced by fibrosis. Pinner points out that the fibrosis of old sarcoids is much coarser than the fibrosis of tuberculosis.

The lungs are involved in most patients with sarcoidosis. The involve-

stage or an
The basic
form seeding
and the lungs
do occur

and in the bases. Instances of regression.

but the most typical resolution is fibrotic replacement of the sarcoid. Not infrequently a linear, non-specific fibrosis eventually develops. A generalized adenopathy is common. The pleura may be the site of sarcoids. Multiple sarcoids of the skin of the face and extremities is of course the most characteristic finding but it is not infallibly present and sarcoidosis cannot be eliminated from a diagnosis on the basis of the absence of skin lesions.



Fig. 171.—A thirteen-year-old white female. This patient had a six-year history of skin lesions on the face, arms and legs. Examinations showed these lesions to be discrete, rounded, soft and purplish, to three mm in diameter. Tuberculin test was negative to 10 mg of O.T. Starch wax lumps were repeatedly negative for tubercle bacilli. Serum albumin was 3.18 gms. γ Serum globulin 3.40 gms. γ . A biopsy of a skin lesion showed a non-necrotic granuloma with typically sarcoid structure. The chest roentgenogram shows fairly discrete, enlarged lymph nodes at each lung with fine nodules extending out to the axilla in the lower half of the right upper lobe (See arrows). Diagnosis: Boeck's sarcoid (pulmonary). (See also Figs. 168, page 32.)

Clinical Symptoms. Sarcoidosis may present extensive involvement with no clinical symptoms at all. When symptoms are present they are often the result of mechanical interference with organ function by the sarcoids, such as intestinal dysfunction because of obstruction. The most common generalized symptoms are loss of weight and strength. A low-

grade fever is sometimes encountered but it is not a striking feature of the disease. When involvement of the lung is extensive dyspnea and other symptoms of impaired function may be found.

Physical Signs In most patients with sarcoidosis the typical skin lesions will be obvious. Where they are not seen small nodules are sometimes noted that are firm to palpation. Enlarged lymph nodes can often be palpated and in many instances the parotid glands will be found to be enlarged also.

The percussion sounds elicited will depend upon the amount of involvement of the lungs. Emphysema is perhaps the most common finding in the physical examination of the chest. If the superior mediastinal and peribronchial lymph nodes are greatly enlarged some dullness over the sternum and interscapular area may be elicited. Under these conditions broncho-vesicular breath sounds may be heard and at times rales.

Roentgen ray Findings—The most characteristic roentgenologic appearance in sarcoidosis is of numerous small soft densities scattered throughout the lung fields. These densities are most numerous in the bases of the lungs. Where densities are not seen in the bases of the lungs, they may be made out upon the basis of roentgen ray studies but such studies are significant when they show densities in the bases of the lungs (see Fig 143 page 282).

The densities seen upon the basis of roentgen ray studies but such studies are significant when they show densities in the bases of the lungs (see Fig 143 page 282) depends upon laboratory roentgenologic appearance of the tubercles have been described under Pathology. Blood changes are not invariable but there is usually increased plasma protein and calcium. The tuberculin reaction is usually negative or weakly positive.

The positive reaction to the injection of a preparation made by maceration of a known sarcoid nodule in saline (Kveim test) is highly suggestive of sarcoidosis. A positive reaction consists of the development of a papule in a period of several weeks. A microscopic section of the papule will show the typical sarcoid nodule. In numerous experiments no case of negative sarcoid involvement gave a positive reaction while in the positive cases nearly all patients reacted positively.

The nature of the lesions in the skin by biopsy and microscopic study is similar to that of the lesions in the lung when the above lesions are not present it is sometimes difficult to make a differential diagnosis between sarcoidosis and tuberculosis. Hodgkin's disease, non-specific fibrosis, pulmonary congestion, metastatic carcinoma and even histoplasmosis.

Tuberculosis—The history of exposure and the clinical signs of cough, hemoptysis, loss of weight, night sweats and fever and the presence of tubercle bacilli in the sputum are definitive differences. In sarcoidosis most patients are in relatively good health although the physical signs and the roentgen ray appearance of the film may be similar. In primary tuberculosis nodulations are noted in the film but the densities are much smaller and more uniform in size and the usual history of prostrating fever and

sweats will usually differentiate the two conditions. In few instances when the cervical lymph nodes are enlarged it may be necessary to perform a biopsy for microscopic study. (See Fig. 140 page 279)

Hodgkin's Disease — Hodgkin's disease, with enlarged lymph nodes both in the axilla and the neck, will give a

Hodgkin's Disease - Hodgkin's disease will frequently show enlarged lymph nodes both in the cervical region and inguinal. In the roentgen ray films one will find enlarged lymph nodes in both discuses. The Pelt Houston type of temperature record itching of the skin loss of weight and even lung abscess formation are common in Hodgkin's disease. The roentgen ray therapeutic test will in many cases melt away the enlarged nodes in Hodgkin's disease and leave the sarcoid nodes unaffected. The crucial test is a biopsy study of available lymph nodes. In many instances a biopsy of the skin lesion may show either sarcoid or Hodgkin's disease when either condition is present. (See Fig 121 page 239)

Angiosarcoma - This condition is the usual result of various conditions particularly the pneumonias which follow measles, although scarlet fever and atypical pneumonias are also associated with this condition.

Acute specific Fibrosis—This condition is the usual result of various conditions particularly the pneumonias which follow measles whooping cough scarlet fever and atypical pneumonia occasionally following chronic atelectasis of the lung and also as a sequel of deep roentgen ray therapy with a history of the preceding conditions and an absence of adenopathy there is little likelihood of confusion in diagnosis.

Pulmonary Congestion—A history of chronic congestive heart failure with decompensation may

There is little likelihood of confusion in diagnosis. A history of chronic cardiac disease, particularly of the chest in chronic passive congestion, the roentgen ray appearance found in sarcoidosis but the routine search for the evidence of metastatic carcinoma. The nodules in the lungs are usually more rounded.

Histoplasmosis—Histoplasmosis is a condition recognized as a rather common cause of pulmonary nodules. The nodules in the lung in metastatic carcinoma are usually more rounded and more regular in size and of course the history of a primary lesion elsewhere is a definite diagnostic confirmation. (See Fig 167, page 322.)

Histioplasmosis—Histioplasmosis is a condition which has been recently recognized as a rather common involvement of the lung with nodulations scattered throughout the lung fields. Hilar nodes are usually not so enlarged in this condition and the nodules are definitely calcified in varying degrees producing highly contrasting shadows in the roentgen ray film. There is little difficulty in differentiating this condition from sarcoidosis. (See Fig 114 page 221)

- BIBLIOGRAPHY
- BIRK MANN A Zur Klinik und Pathologie der Boeckseln in Lungenerkrankungen. Beitr
z Klin d Tuberk 92 581 1917
BERNSTEIN S S and SUSMAN M L The rare manifestations of sarcoidosis. Radiol
44 37 1917
BOECK C Multiple benign sarcoid of the skin. J Cut & Gen I Dis 17 547 1899
DANFORTH A Sarcoid reaction in Boeck's sarcoid. Acta med Scandinavica 114 143 1917
GARLAND F H Pulmonary sarcoidosis: early diagnosis and findings. Radiol 48 333
1917
HARRIS G T Generalized sarcoidosis of Boeck's clinical review of 11 cases, with
notes on etiology and etiological factors. Arch Int Med 65 1013, 1910
KING D S Sarcoid disease as revealed in chest roentgenogram. Am J Roentgenol
45, 92 1917
KRYM A Preliminary report on new and specific cutaneous reaction in Boeck's sar
coid. Arch Int Med 9 163 1917
KRYM J Therapeutische Ergebnisse histologische Bilder bei Hauttuberkulose und deren
Bedeutung im allgemeinen Mikroskop (Boeck) Arch f Derm 100, 375, 1910

- MALRICE, L., and HINKLE, C. L. Sarcoidosis—report of two cases, Bull U S Army M Dept 6, 617, 1946
- PINNEY, M. Noncaseating tuberculosis, Am Rev Tuberculosis, Am Rev Tuberc 37, 690, 1938
- Pulmonary Tuberculosis in the Adult, Springfield, Illinois, Charles C Thomas, 1945, p. 312
- REISNER, I. Disease
- RICH, A. R. 1944, p. 100
- and P. M. Sarcoidosis—noncaseating granulomatous reaction of
-
- TÖRNELL, E. Is sarcoidosis a fungoid disease? Acta tuberc Scandinav 20, 212, 1946
- WARFVINGE, L. E. Skin reaction caused by killed tubercle bacilli in lymphogranulomatosis benigna, Acta tuberc Scandinav 19, 126, 1945

INDEX

(Note: Main entries are indicated by bold face numerals.)

A

[illegible]

133

- Bronchoscope for sputum study, 17
 in bronchial obstruction, 27
 in bronchiectasis, 169
 Bronchoscopy in bronchiolitis, 150
 in obstructive atelectasis, 27
 of carcinoma of the lung, 322
 Bronchopneumonia, 169
 and edema of lung, 170
 and mediastinitis, 104
 bronchoscopy in, 172
 clinical symptoms in, 171
 differential diagnosis, 169
 lobar pneumonia, 172
 putrid lung abscess, 173
 tuberculosis, 173
 empyema in, 76
 etiology in, 169
 in pneumoconiosis, 218
 laboratory findings in, 172
 pathology in, 169
 physical signs in, 171
 recurrent attacks of hypoid pneumonia in, 186
 roentgen ray findings in, 172
 Bullae, emphysematous, 24, 256
 Bullet in lung and calcified pleura, 67
 Byssinosis, 215

C

CALCIFICATION OF ADVENTITIOUS TISSUE

C

- Cancer of the lung *See* Carcinoma of the lung
Candida albicans in mouth, 234
 in pulmonary mycoses, 226
 Carcinoma cells in effusions, 22
 etiology of, 316
 in mediastinitis, 100
 laboratory tests in, 323
 metastasis with effusions in, 21
 of pleura, 59
 of the lung, 81, 163, 182, 263, 316, 316
 clinical symptoms of, 320
 differential diagnosis of, 324
 atelectasis, 327
 benign growths, 328
 calcified lesions in the lung, 327
 fungus disease, 324
 lung abscess, 324
 pneumonia, 325
 sarcoidosis, 326
 silicosis, 326
 tuberculosis, 324
 pathology of, 317
 physical signs in, 319
 producing atelectasis, 235
 roentgen ray findings in, 321
 stain studies of sputum in, 183
 thoracentesis in, 323
 thoracoscopy in, 323
 with serofibrinous pleurisy, 65, 66
 Cardio-inhibitory reflex, 25

- Cardiospasm, 112, 118
 Cartilage deformities of, 39
 Caseous nodule, 61, 85
 Casom intradermal test in hydatid cysts, 261
 C
 rupture of, 204
 Cells epithelioid in pneumoconiosis, 217
 Cervical lymph nodes in Hodgkin's disease, 238
 rib, 39, 45-47, 46
 anesthesia in, 45
 as rudimentary first rib, 45
 brachial plexus in, 45
 clinical symptoms of, 45
 differential diagnosis of, 46
 apical lung tumors in, 46
 burstis in, 46
 syndrome of the humerus and clavicle, 47
 etiology of, 45
 Horner's syndrome in, 45
 paresthesia in, 45
 pathology of, 45
 physical signs in, 45
 roentgen-ray findings in, 46
 Cholecosts, 215

- empyema necessitatus, 52
 hydatid cysts of pleura, 51
 lung hernia, 52
 Paget's disease of the bone, 52
 subscapular abscess, 52
 etiology of, 48
 laboratory findings in, 52
 mycotic infections of, 44
 pathology of, 48
 physical signs in, 49
 Recklinghausen's disease of, 51
 roentgen ray findings in, 50
 tumors of, 48

Chondro

- Empyema pneumococcal clinical symptoms of 77
 pockets in pulmonary mycoses 233
 pulmonary abscess 204 207
 putrid 76 83 84
 roentgen ray findings in 77
 serofibrinous pleurisy in 64
 tuberculous 72 86 85-87
 allergy in 85
 clinical symptoms of 85
 differential diagnosis of See Sero
 fibrinous pleurisy
 etiology of 85
 laboratory findings in 246
 pathology of 244
- See Sero
 vst
- Eosinophila in hydatid cyst of lung 261
 262
 Eosinophilic infiltrations of the lung 19
 pneumonia See Pneumonia eosino
- G
- GARDNER's experiments in pneumoconiosis 218
- H
- HEART and lungs and blood vessels 146
 Hemoptysis and bronchiectasis 150
 fatal in bronchiectasis 153
 Hemorrhage of the lung in pulmonary mycoses 221
- F
- physical signs in 240
 roentgen ray findings in 240
 with mediastinal tumor and lymphoma
- encapsulated empyema 261
 non parasitic cyst 264
 pulmonary tuberculosis 264
 ruptured hydatid cyst of the liver 265
 thoracic tumors 262
 tuberculous pleurisy 263
 etiology of 258
 laboratory diagnosis in 261
 pathology of 258
 physical signs in 259
 roentgen ray findings in 260
 of the pleura 51
- Fungi in empyema 76
 in sputum 17
 Fungus infection in serofibrinous pleurisy 68
 Funnel chest 39

Hydrogen moper a l m u t l pulmonary l u g d f f e r t u a l j k n o w s d l r e n t e e
 t l e c u l a u
 H i r o t l r a y a n l a m t a w t l o a n a n
 t m o r 74 5

I

I n f e r e n c e a l p o m e n a e l a l e v n p l o m
 l n g n a l l e r n a n n p e e t
 e t o l g y o f 127
 I n o r t a l n g i n a j k n a r o f i l e u u
 p l n y 68
 I n p e c t u n n l a g n a 10-13
 I n t e r t a l l l o a d v e a l p e t r e f n
 p n a o l l r a x a d
 n r e v n n f r o m o f 14
 I n t l l i r e f f u n s
 e m y n a e x l a t o r y p e r a t u n n 81
 m e t g n r a y f l i n g n 177
 I n a t l r a l j n u m l a a p l n n a r y
 t l e r e l g a l a p e r t s f 28J
 I n l m l r n l s e c i j l
 l o d e l o l n r o u n t e n o g r a p h 33 5 e s
 77 0 108 1 a 707 370

K

k a v e r e n t r o f n a f l i n o j l r n a
 (1
 p n n a u t 61 n o a j l n e l
 k u n d e k a p l a n o m n e p n t n n
 p n m l r a x 3
 A p l u s n t l o a c d f r m t u s 3470

L

L a b o r a t o r y l a g u e s p e r f l a u
 e n s e
 m t h o d n d a g n a l
 l a n n a g a p l 33
 l a n g l a n a e l t l e r e t n 717
 l e a k t e o n t a l b a r j m n a j m
 l p l p n n a s e l n n l p o u l
 l p m j p 5 106
 l a p j a g n a l j l u n e 181
 l a n n a g a f m e l a n u m 106
 l t n a g n n e r f r u n p u
 61
 l a r a l n a n e n f r u n p l u n e l
 l a e t n y n l m u n a r y t e r a l 301
 l e a l e z e p p a l n f e l n n
 t n l e r e h a r t a n a l
 l a m l a k l a n e l a e
 l o n l n t l u n e n g 3
 l a n g a l o o P u l m a r y
 a c q u i r i n g t y f 131
 e n e n l i n g l n t a
 t l e x 1
 a n y o f 141
 n o d l a r t a l l a l a e l j
 a t l e c t a o f a n t h l n t h o r a x
 l e n g t n r e f l e 310
 l i t a n t e a l e s l p l r a
 e n n m a o f a n n a
 n a l a n j m f 711
 e n p e n a t e n n i n n a f 4
 l f f e n t a l l g n o w f 313
 a n e n e f t l e n g 314

calcinosis alveolar, 314
 carcinoma 313
 empyema 314
 tuberculo 313
 each noceros cyst of 16
 ed m o f a n l l e n c h o j n e r n o n a l o
 t o l o g y o f 310
 e x a n n a t i o n o f 14
 h e m o r r h a g e n p l m o n a r y m y c o s e a n y
 l e n a o f t h e c l e s t w a d
 l y d a t e c y s t o f s e l y d a t e d c y t o f
 t h e l u n g
 l a b o r a t o r y t e s t n 313
 l e a n o n (n l o d g k n e d w a y 238
 m e l n e c u s l e a w e s o f 147
 n y e i n s o f S e e P u l m o n a r y m y c o s e
 n p a r a s t e c y s t o f 71
 p a r t l e o l p e f 68
 t o l o g y o f 310
 t e x e a l e g n n 317
 t u b e r c u l a r f i n d i n g s n 317
 " B o n l o l l
 t i p r a t n a m y k a l o w n 70
 t n u r e f 57 310 a l o o (a c i n m a
 u r i n e a n a l y n 70
 l m p l a u d t p e f 710
 n l g l n n p e e t n 13
 e r a l n H a l g k n l e a y 238
 l p a r t i n j l f 10
 t a l e r t r o l a l l e y a t n f m a d
 n e t 100
 l n t m a n H a l g k n a H a l g k n
 d e n a
 l m j a n e n 48 f
 H a l g k n d e n a 238
 l n g u l n n e t e n l k 33

M

M a d r a m t e r l e s
 M n t e r l i n g n e t f l i n g
 M a s s e p n m n a l
 M e l a l a l e n 22
 M i n t n t 100
 l a l a m j n f 101
 l f f e n t a l l g n f 101
 u j m a 101
 p r a j a l a l a w e s 11
 j n n 101
 t n s 103
 t o f 100
 l l e r r a t t n 17
 p o t a l g y f 106
 p l a l g n 101
 n a n g e n s f i n l i n g n 111
 M i n u n a l a w e s f 100 101 10.
 a n y n f
 l o n e d f m f 10
 d f f e n t l o g n s 112
 a l a w e s 117
 l l t n f t h e r e p l a g u x 117
 l e f f r o u r u r a f 111
 e k 70 f 106
 l e r e c e s t n 111
 l n j l e x e m a f
 n n f i l m m f e
 p a t d a g f 116

- Mediastinum physical signs in 107
 roentgen ray findings in 108
 sarcoma of 115
 section through 97
 spontaneous pneumothorax 92
 teratoma of 106 113 114
 tumors of the 104
 with Hodgkin's disease and hydrothorax 76
- Oil aspiration in hydropneumonia 184
 extracted from lungs at autopsy in hydropneumonia 187
- Ossification patches of in hydatid cysts 201
- and
- 41
- tumor See
- Papaniolou technique of distinguishing
- coccidioidomycosis 227
 cryptococcosis 227
 histoplasmosis 226
 moniliasis 226
 North American blastomycosis 227
 sporotrichosis 227
 streptothricosis 228
- Mycotic infections of chest wall 44
- Myxomas of mediastinum 106
- N
- NASAL SINUSES chronic infection of and
 bronchiectasis 152
- spontaneous pneumothorax 255
 tuberculous cavities 255
- etiology of 250
 laboratory findings in 254
 pathology of 250
 physical signs in 251
 roentgen ray findings in 252
 thoracentesis in 254
- Normal appearances of chest in roentgenology 36
- Neurofibroma of chest wall 51
 of intercostal nerves 12 14 48
 of mediastinum 106 109
- Pel-Ebstein fever chart in Hodgkin's disease 240
- Photofluorography in roentgenographic surveys 36
- Plotometer Morgan in chest survey examinations 36
- Pleural crush in pulmonary tuberculosis 292
- Phrenicotomy in pulmonary tuberculosis 292
- Physical signs in diagnosis of aortic aneurysm for interpretation 16
 See Specific disease

- 17 cal f d an i l l t n i n g 5
 fibro s f i n e m y o m a t o n i n g n r a
 f i n i n g s n 77
 m m o b i l i z a t i o n o f i n a c u t e f i b r i n u s
 p l u r i y, 61
 n t u b e r c u l o s e m p y e m a 8 a
 n f e c t i o n o f i n p n e u m o t h o r a x 2
 m e l l i n e o s d e n s e o f 5 a
 s f a c e o f e a s t n g n o d u l s o n i a c u t e
 f i b r i n o s p l n y 61
 t u m o r s o f 5 a
 P h u l c a v i t y t h o r a c o c p o f 27
 c y s t a s
 e f f n e m p l e a t i n g m l a t n a l t m e r
 107
 t o n i g n l e g y i n 33
 s e r o s w a l l u l l e r a l s e e 137
 e x u l t n e r e f f i n s p l n c s
 f i l l o d y c l o t n s y
 f i n e a n d l r n e h t a 153
 f i t n n s l i f f i n a l c s 137
 n v l e a n t n f l a g h n e d w a s e 140
 j r x r e n n e r n t o f n f u m e r
 t l r a x 27
 j r x r e n n e r n t l r a x 2
 n t l r a c i n t s 2
 c l i n o 50
 a l k i n i n m o t h a x 2
 t l k n g i n p l m n r y n o s e s 233
 n t l r a c i n t s 2
 P h n a l c a l c l
 a c t f i b r n s e A t f i b r n
 i l l r a l n n l t n t 101
 m e f i r s n f i b r n f l n
 P r i n t l t f l m n a r y a l s e e 204
 i n p n e m e n s
 P h m u c o e r a l m p n s c l n l n j
 t n s o f 77
 n f t s a n i a t f i b r n f l
 c l
 P r e m u c o s 215
 l e n g n 21
 c l n c a l e n t n f 211
 l f f n t a l l a g n f
 f n g s n f t n 21
 n n a l l l o g 21
 t l a r e 21
 e t l r f 215
 C a n i n e e x p e r i m e n t n 218
 l a l a t o r y a t l s e
 p a t l k g s o f 216
 p h c a l s g n n 21
 m o n t g r a v e x m a t e n 21
 P r n u l t h 14
 P r n u s s B r e n l l m s
 a t l e t a n s
 l a g n e t p n e r n u l t r a x s
 n p l 193
 p n s m e a
 i n c a l m p t n f l r
 l f f r e n t l i a g n f n
 e t u d g s o f l r
 l f e r s t f i n i n g n l r
 p a t h o l o g f l r
 p l c a l g n n l r
 n o n t g e s l g s f i n g n l r
 l e t u l r 170
 P n m n s l p o s t a t e 16
 n f f n z a l c l a s n j t m f l l
 e t i o l o g y o f l r
 l p o l 184
 a c u t e 184
 a n d b r o n c h e c t a s 185 186
 t r o n c h o p n e m u n a r e c u r r e n t a t t a c k s
 o f 186
 b r o n l s c o p y n 184
 l n c a l s m i t m o f 186
 l f f r e n t a l d i a g n s f l r
 b r o n c h e c t a l l
 c a r e n n a 180
 f n g i l s a s l l
 l l m e r y t l a r e l 191
 s l e o 184
 e t l r f 184
 l a b e r a t r y l a g n f n a p t n 17
 t n s n 188
 m n e r o p e s e e t n n f
 l x t r a t e d f r o m l k a t a p
 n a c a s e o f l r
 j a t l l o g f 184
 p l c a l s g n n 184
 r o s t e n r a f i n i n g s n 186
 l l 175
 a l r u s i n m n s 133
 l n c a l s m i t m o f 177
 l f f r e n t a l l k n l
 a c t o f i b r n a l m e n f l m o
 p l 180
 a t l e t a 180
 a p p l e t 18
 n e r f i l l n g 184
 g e s t l u r t l s n a n i e r m
 n r y e e l n 180
 m p n r 181
 p l o w e r n a r t 171
 n l m r n
 t l l o g f 17
 t l s a r y f i n g n 178
 l t h l g f l l
 n e t x n r y f i n i n g s l r
 l t a l l f t 78
 p l t r a t w t l l i f f n a l s e e
 137
 p e t m n 17
 n l l a l 192
 l n a l s m i t n l l
 l f f r e n t a l l g n o s o f l r
 l t u l p n n o n s l r
 l a g n e n t r o m e 18
 t l a c e n 184
 t u l a g f l
 l a b e r a t r y f i n i n g s n l r
 p a t l k g s f 183
 p l c a l g n n l r
 l r e s t l l p a t m r y l l m n s
 213
 n e t e n l o g f i n g n l r
 P n n u m e t m n p n n u r y t l e c
 l a s 201
 P r n o n a n i l m u l t e t a l 3
 P n n u m e t n t l e c a l s e n t e m s 51
 P n n u m e t n e u m a t f e c a l 9
 n t n u r y t u e c a l s e c
 P n n u t l r a t a t a l s
 l a g n e t 201
 n c o l l e c t i o n f l l u s e

- Pneumothorax, in locating tumor, 59
 in pneumonia, 78
 in pulmonary abscess, 207
 cysts, 205
 tuberculosis, 293
 in tumors of pleura, 27
 machines, Singer portable, 100 cc capacity, 26
 1000 cc capacity, 20
 method of, 27
 pleural pressures in, 25
 puncture of, intercostal blood vessel in, 59
 recurrent 88
 spontaneous 58
 acquired cysts of lung resembling, 93
 caused by puncture by fractured rib, 89
 caused by tearing of adhesion, 89
 causing mixed tuberculous empyema, 85
- P
 Prothrombin, 20
 Pruritis, generalized, in Hodgkin's disease, 240
- Pneumonia *See also* Pneumonia virus
 clinical symptoms of 194
 etiology of, 192
- Pulmonary *See also* Lung
 abscess, 117, 202
 bronchoscopy in, 208
 clinical symptoms of, 204
 differential diagnosis of, 209
 bronchiectasis, 213
 bronchobiliary fistula, 213
 bronchogenic cysts, 210
 carcinoma of the lung 209
 empyema, 213
 fungus disease 210
 subphrenic abscess, 213
 suppurative peribronchial nodes, 211
 tuberculosis, 209
 etiology of, 212
- Pulmonary, tuberculosis, clinical symptoms in, 270
 differential diagnosis of, 277
 atypical pneumonia, 282
 bronchiectasis, 278
 bronchopneumonia, 281
 lung abscess, 280
 mycosis, 279
 sarcoidosis 282
 silicosis, 278
 the pneumonia, 281
 tumors of the lung 280
 etiology of, 266
 extrapleural pneumothorax in, 294
 intrapleural pneumolysis in, 289
 laboratory tests in, 275
 lobectomy in, 301
 medical aspects of, 266
 olothorax in 291
 open pneumolysis in, 289
 pathology of, 267
 phrenic crush in, 292
 phrenicectomy in, 292
 physical signs in, 270
 plombage in, 296
 pneumonectomy in, 301
 pneumoperitoneum in, 301
 roentgen-ray findings in, 271
 Schilling's test in, 19
 surgical aspects of, 285
 thoracoplasty in, 298
 with adhesions, cavitations and fluid level, 67
- R
- Rocky Mountain fever in virus pneumonia, 176
 See Specific di-
- trial, in bronchopneumonia
 iodized oil in, 33, 77
 tumors of pleura, 59

- Rupture of the cavity of pulmonary abscess 315 n of et r s cu ng s l j l ren at
 204
 R t sp l m n l l ar p monia 177
 S
 Saco box s 330
 clinical sympt ms of 331
 differential diagno s of 332
 h toplassmo s 333
 blood n s l case 333
 n tartaric acid n ma 333
 non-specific filro 333
 p l n nary engest n 333
 tularemia 33
 tw l g s of 330
 laboratory findings n 332
 j t l l g y f 330
 pl calx g s f 332
 roentgen ray find ngs n 332
 s roo l (f l n nary) 331
 s roo s grant of j l n 9
 n n p e t n 12
 f t l el st wall 48
 of m s l a t n n 112
 f t l r h s 48
 s ap l a t u l a r u l f e t o n f 43
 s e l o n n l y l a t l e y s t a 2)
 s e l o n n l y l e y s t a 41
 s e d m n l a t n r a l n n f e t u t l a r
 l u s 20
 of erythrasma 11
 s e h l e r s e l l u n y a l e l m a 77
 n w h e r e n n r f t l l n g t 6
 s e r o f f n n n m o f i l r o p l n e s
 s l e k a n a s y l a c t e s e l r s l e k
 s l n e 21
 s e s 215 219 220
 a n t i b i o t i c s 12
 h e a r d 216
 p l t l l n g a p l n 36
 n t l t l e r e l s f
 s n g r n p u a t n n t l d o f n p r e n g
 n w l z f 13 3
 e l a r t f e n t q n t a t n f p l y e n l e g n s
 m t h d f f e t l o r a c o n p y 27
 p m e m o t o r a c s 20
 p m e m u l t r a c n a c n e 100 c e c a p a c i t y
 25 27
 1000 c e c a p a c i t y 26 27
 p e t a l l m a c l n e f o r p n u m e t o r a c
 n e r a l t l r a c o n p y 28 29
 n l e l e g n g n t l e t l e r l u n f e
 u n t o n o f t h e t l r a c e a g e 43
 f o r m a t i o n o f t h e e r e c t w a l l n m e t e n
 f e t u s n n t a n g r a m f 44
 n a s a l e h n e n f e t u n f a f l l n l n
 e r i a 132
 k e l t l d f r m t s o f t h o r a c e a g e 39
 s k n t u p s o f 210
 s m e g m a i n u r i n e a n a l s 20
 s e n d a p p r a i s a l o f n e x a m i n a t i o n 14
 i n t e r p r e t a t i o n o f 16
 s p e r m i n a c o l l e c t i o n f f e t l e r a t n
 a n a l s 17
 s y l h o g u n l n s p e n t a c o s p e r m e n
 t h o r a x 21
 S y l e n o f e t r s c u n g s l j l r e n a t
 s e e s 136
 s p r n t a n s p n m u t o r a c s e l n
 s p r o t n e l n s 12 227
 s p o a t r i c h u n s e h e n k
 t r e h 227
 n p u l m o n a r y m y c o s s 227
 s p o t m o n t e n o g r a m 33
 s p u t u m b l o o d t n g d n l p l n m e
 186
 n d g n o 17
 s t a n s t u l e s f e n c i n n a s
 s t m m d f o r m t s o f 49
 s t r u c t u r e e x a m i n a t i o n
 g r a p h y 36
 r o n t g e n a g s 33
 r o n t g e n g r a m 36
 s t n a h l e r e s s l j l n
 136
 f e c e s e x a m i n a t i o n p 20
 n a h t l n f l l
 s t r u c t u r e 33
 s n t t l n e r 224
 s u b a p l a n g m a t a l s
 s l n n l e c e s
 p h r e n a b s c e s s 136
 e l n o l a m y n f 13
 i f f r n t l d g n f 13
 e l n a b s c e s s f 13
 13
 p e n 13
 l k a l e s 3
 l u n a 13
 l l n l r x 13
 t l l g s f 13
 l l e r a n f l n g n 134
 f t l l g s f 13
 j l a l g n f 13
 r o n t g e n a f l l g s 13
 s b l n n t n n n e r f l e n t n
 64
 s l o c a l y a a c e n t l t t w a l l s
 s l l n s a t m e n 40
 s j a n n 60
 s p l n l n l m y n a
 s r o l a s p e t f l l m n a r t l e r e l
 f t l e r l 235 s e e s e t l e r e l
 p u l m o n a r y
 s r y a n e n g r a p h y 36
 s m j a n t f l l n
 s l l a n t m a t a l n 100
 s n n p a n l 2
 s p l l t l s n f t l t r e a g e 41
 T
 T a n g e n t a l l y r a l l e r l t n m e n t
 g r a p h y 33
 T e n a n p n n l e s s s
 T e r a m a f m e d a t u m 100 114 114
 T h r a c e a g e 11 33 2
 T h r a c i n e s
 l a g n e t s
 T h a x f n t u w f
 l a t r a s w o f
 t r a n s m e m b r a n e n g h

Thoracoplasty in pulmonary

298

in tuberculous empyema, 85

Tomography, 33

Torula histolytica, causing cryptococcosis, 227

Tracheo-bronchial tree, 33, 67, 141

anatomy of, 141

miscellaneous diseases of 147

Transudate effusions, 21

Trichoblast, 11

Tubercle bacilli, in sputum, 17

stomach wash for detection of, 19

Tuberculosis and acute fibrinous pleurisy, 61

and bronchiectasis, 152

^{41, 42}
Tumors See under Tumors in specific conditions

U

URINE, laboratory analysis of, 20, 43

Urticaria skin, in bronchography, 35

W

WESTERGEN method, rate of sedimentation of erythrocytes in, 20

Whisper sounds, in auscultation, 15, 16

Wintrobe method, rate of sedimentation of erythrocytes in, 20

pneumonia, 241

pulmonary, 102, 173-226 See also Pulmonary tuberculosis

